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of the
American Veterinary Medical Association

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(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

Col. R. J. Foster, Pres., Washington, D. C. — M. Jacob, Treas., Knoxville, Tenn.

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CONTRAST

Several months ago, upon being called to the telephone, we found ourselves engaged in conversation with the credit department of one of Chicago's giant financial institutions. The official on the other end of the line stated that a local veterinarian had made application for a loan. In the process of investigating the applicant, the bank called up the American Veterinary Medical Association to get certain information. It seemed to be a perfectly logical procedure, in view of the fact that the prospective borrower was a veterinarian. We stated that we had a record of the man in question, that he was a graduate of a certain veterinary college, of a certain year, and that he was licensed to practice in the state. However, our information was limited to these facts, as the party in question was not a member of the A. V. M. A. Well, was he a member of the local association? No, he was not. Well, what about his state association? Again the answer was no. As the conversation ended rather abruptly at that point, we do not know whether the veterinarian got the loan or not. But we might be able to make a guess.

A couple of weeks later, upon answering a telephone call, we heard a party at the other end of the line say that he was speak-

ing for the credit manager of a large manufacturing concern in Chicago. It appears that the company had received an order from a veterinarian in California who had not had an account with the company previously. The question of his credit came up, as the order involved quite an amount. The order had been written on the professional letterhead of the veterinarian and this fact had suggested calling the A. V. M. A. To make a long story short, we were glad to report that the veterinarian in question was a member of the A. V. M. A. and had been such for a good many years. We gave some additional information, all of which seemed to impress the credit man favorably, and were told that the order would be shipped at once on open account and without further ado. What a difference!

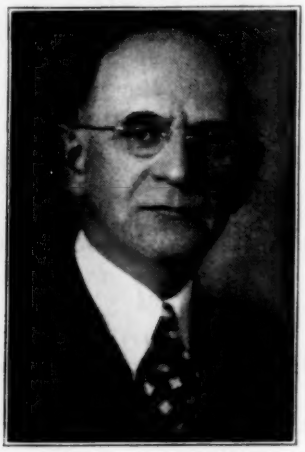
YOUR INVITATION

On the opposite page you will find your invitation to attend the 74th annual meeting of the A. V. M. A. in Omaha, Nebraska, August 16-17-18-19-20. In one way or another this invitation is being placed before every graduate veterinarian in the United States and Canada, whether he or she is a member of the A. V. M. A. or not. Although the convention is being held in Omaha, it is not strictly an Omaha affair, as the veterinarians constituting the Committee on Local Arrangements have been drawn from a much larger territory, extending a hundred miles or more into Nebraska and Iowa. The various subcommittees, under the general direction of Dr. W. T. Spencer, of Omaha, have been hard at work for six months laying the plans for taking care of the hundreds of veterinarians and the members of their families who will flock to the Nebraska metropolis during the third week in August.

Recent conventions of the A. V. M. A. have set a very high standard of entertainment, and naturally it is the desire of each succeeding city in which we meet to equal or surpass this standard, something that is becoming increasingly difficult each year, as attendance figures continue to climb. Just remember that there were approximately 1,850 registrations at Columbus last year. Compare this figure with 134, which represented the total attendance at the first Omaha convention, back in 1898, with the Trans-Mississippi Exposition as an added attraction; or just ten years ago, when we met in Philadelphia, with a total attendance of only about 800. It has become a real job to plan an A. V. M. A. convention, but Dr. Spencer and his corps of

The Officers
of the
American Veterinary
Medical Association
and
The Members
of the
Committee on
Local Arrangements
invite every veterinarian in America
to attend the
Seventy-Fourth
Annual Meeting
Tuesday, August seventeenth
to
Friday, August twentieth
nineteen hundred thirty-seven
Omaha

able lieutenants appear to have the situation well in hand as this is written, ten weeks before the meeting. Plan now to be in Omaha the third week in August. The program proper will start with the opening session, Tuesday morning, August 17.



DR. W. T. SPENCER
Chairman of Committee on
Local Arrangements

STATE ASSOCIATIONS ACT ON TRANSFERABLE MEMBERSHIP

No less than a dozen state associations have taken favorable action on the suggestion made in the JOURNAL (January, 1936, p. 1, and December, 1936, p. 629) that veterinarians in the employ of the federal government, particularly those in the Veterinary Corps of the Regular Army and the Bureau of Animal Industry, be offered the opportunity of securing what would amount to a transferable membership in our state associations. A veterinarian in the Army or the B. A. I., subject to transfer from one state to another at any time, has faced the necessity of paying a membership fee whenever he has joined a state association, with few exceptions. With the general adoption of the suggested plan, this will no longer be necessary. Two variations of the plan have been considered. One makes membership in the A. V. M. A. a prerequisite. The other requires membership in at least one state association. In either case the customary membership fee is waived. States which have reported favorable action to date are:

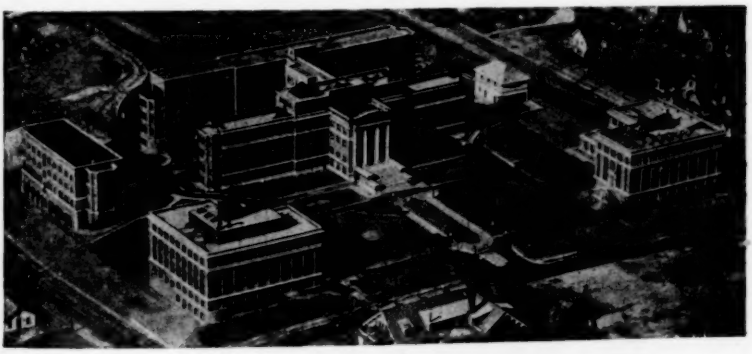
Colorado	Louisiana	Ohio
Connecticut	Maine	South Carolina
Illinois	Maryland	Virginia
Kansas	Michigan	Wyoming

It is possible that other state associations have taken action but have not reported, either to the secretary of the A. V. M. A. or to Dr. F. A. Imler, secretary of the National Association of Bureau of Animal Industry Veterinarians, 19 Federal Building, Kansas City, Kan.

EXECUTIVE BOARD ELECTIONS

The regular elections now in progress in Executive Board Districts 1 (Canada) and 9 (New York and the New England states) will come to a close on June 21. These elections will decide the two members who will represent their respective districts on the A. V. M. A. Executive Board for the five-year terms beginning at the close of the meeting in Omaha. In each district the present member of the Board has been nominated for reëlection. Dr. A. E. Cameron, in District 1, and Dr. H. W. Jakeman, in District 9. The voting has been unusually heavy, particularly in District 9. One possible reason for this may be found in the fact that eight candidates appear on the ballot for that district, the result of a five-way tie for fourth place in the primary election. There are approximately 500 members in District 9.

The greatest hatred, like the greatest virtue and the worst dogs, is silent.—Jean Paul Richter.



UNIVERSITY OF NEBRASKA MEDICAL SCHOOL AT OMAHA

NOTES ON THE OMAHA CONVENTION OF 1898

The number of members in attendance was 43.

There were 23 ladies present from seven states.

Fourteen applicants for membership were at the meeting.

Forty new members were enrolled and two former members were reinstated.

As a result of the new members admitted, the total membership was increased to 318.

Non-member veterinarians registered reached a total of 49 and five other visitors brought the total attendance up to 134.

Two members enrolled at Omaha subsequently were elected to the presidency: Dr. John R. Mohler and Dr. Charles E. Cotton. Another member-elect, Dr. John J. Repp, later served as secretary.

Among the 43 members present were 13 whose names now appear in the list of past presidents of the A. V. M. A. They are: Bell, Brenton, Cary, Clement, Hoskins, Knowles, Law, Lowe, Merillat, Pearson, Salmon, Stewart and Williams (W. L.).

Of the six officers who served at the 1898 meeting, only one is now living, Dr. A. T. Peters, then of Lincoln, Neb., now of Peoria, Ill., who was Third Vice-President. Of the six officers elected to serve for the year 1898-1899, all have passed to the Great Beyond.

Perhaps the most interesting feature in connection with the first Omaha meeting was the surgical clinic. On several days the morning hours were given over to the clinical demonstrations arranged through the courtesy of the Committee on Local Arrangements. This feature marked the initiation of the clinic as a part of the annual convention program. The proceedings of the meeting record six operations as having been performed, five on horses and one on a bitch (ovariotomy).

In looking over the membership list published in connection with the proceedings of the 1898 meeting, it is interesting to note where some of the present members were located 39 years ago. Dr. Tait Butler was at Starkville, Miss. Dr. Paul Fischer was at Manhattan, Kan. We find Dr. J. I. Gibson located at Denison, Iowa. Dr. Chas. H. Higgins was listed at Fitchburg, Mass. Dr. N. S. Mayo was at the Agricultural College at Storrs, Conn. Dr. John R. Mohler was stationed at the Plankington Packing Company, Milwaukee, Wis. The address of Dr. E. M. Nighbert was given as Mount Sterling, Ill. Dr. R. J. Stanclift was at Americus, Ga.

ARMY LEGISLATION

On April 29, 1937, Senator Royal S. Copeland, of New York, introduced a bill (S. 2311) entitled, "To provide for adequate medical and dental service for individuals of the Military Establishment of the United States." This bill was read twice and referred to the Committee on Military Affairs. Section 1 of the bill reads as follows:

> Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That section 10 of the Act entitled "An Act for making further and more effective provision for the national defense, and for other purposes," approved June 3, 1916, be amended by providing that on and after July 1, 1937, there shall be officers as now authorized by law, except that there shall be * * * seven assistants to the Surgeon General with the rank of brigadier general, one of whom shall be an officer of the Dental Corps and one of the Veterinary Corps, and * * * one thousand one hundred and eighty-three officers of the Medical Corps and two hundred and fifty-eight officers of the Dental Corps: Provided further, That vacancies created by this Act shall be filled by appointment of fifty medical officers and twenty-five dental officers in each of the fiscal years 1938, 1939 and 1940: Provided further, That the total number of officers authorized for the Army is hereby increased by two hundred and twenty-five.

It will be noted that if this bill is passed without any change, it will give the Veterinary Corps a brigadier general. The value of this higher rank for an officer of the Veterinary Corps can not be overestimated and members of the veterinary profession are being asked to write their senators and representatives at Washington urging their support of the measure. It is suggested that each veterinarian write a letter to Senator Copeland, thanking him for supporting this legislation. If you have not already done so, write letters to your Congressmen and Senator Copeland along the lines suggested. Act now. Do not put it off.

APPLICATIONS FOR MEMBERSHIP

(See January, 1937, JOURNAL)

FIRST LISTING

AKIN, JOE E. 703 Nebraska St., Sioux City, Iowa
D. V. M., Iowa State College, 1934
Vouchers: S. S. Gibson and T. W. Munce.

ANDERSON, ROLLAND Box 175, Worland, Wyo.
D. V. M., Colorado State College, 1935
Vouchers: H. D. Port and I. E. Newsom.

ANSTEY, JOHN A. Massena, Iowa
D. V. M., Iowa State College, 1905
Vouchers: Paul L. Matthews and W. T. Spencer.

BOND, THOMAS L. Cumberland, Iowa
D. V. M., Kansas State College, 1933
Vouchers: Paul L. Matthews and W. T. Spencer.

BOONE, CHARLES W. R. 2, Roanoke, Va.
D. V. M., Cincinnati Veterinary College, 1918
Vouchers: W. P. Weaver and Edward E. Robinson.

BUNKER, JOHN W. Winterset, Iowa
M. D. C., Chicago Veterinary College, 1902
Vouchers: Paul L. Matthews and Rob't. D. Wall.

CAMPBELL, HARRY C. 2300 Locust St., Philadelphia, Pa.
B. S., Pennsylvania State College, 1900
V. M. D., University of Pennsylvania, 1902
M. D., Temple University, 1907
Vouchers: Israel Live and Wm. J. Lentz.

CRALL, WILLIAM W. Pioneer, Ohio
D. V. M., Chicago Veterinary College, 1919
Vouchers: John H. Lenfestey and E. C. W. Schubel.

DENHAM, WILLIAM B. Cumberland, Iowa
D. V. M., Kansas City Veterinary College, 1914
Vouchers: Paul L. Matthews and W. T. Spencer.

DITMAN, NORAN L. Winside, Neb.
D. V. M., Iowa State College, 1932
Vouchers: Paul L. Matthews and S. S. Gibson.

GARRETT, HARLEY U. Saint Charles, Iowa
D. V. M., Kansas City Veterinary College, 1914
Vouchers: Paul L. Matthews and Rob't. D. Wall.

GEHRMAN, FREDERICK W. 5324 Lyndale Ave. S., Minneapolis, Minn.
B. V. Sc., Ontario Veterinary College, 1937
Vouchers: Joab P. Foster and Vincent J. Robinson.

HAGE, DENNIE M. Ida, Mich.
D. V. M., Grand Rapids Veterinary College, 1914
Vouchers: E. C. W. Schubel and H. H. Fairbank.

HALVER, DALE L. Shakopee, Minn.
D. V. M., Iowa State College, 1922
Vouchers: Joab P. Foster and Vincent J. Robinson.

HANSEN, FREDERIC W. Pelican Rapids, Minn.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: L. E. Stanton and C. P. Fitch.

HODDER, CHARLES P. 19426 Troy Place, Detroit, Mich.
D. V. M., Michigan State College, 1936
Vouchers: L. H. La Fond and S. R. Elko.

HUNSBERGER, LT. RAY S. Philippine Dept., U. S. Army, Manila, P. I.
D. V. M., Cornell University, 1934
Vouchers: Lt. Col. Clell B. Perkins and Capt. S. M. Nevin.

JEFFERS, BOYD 355 Walnut St., Lexington, Ky.
D. V. M., Indiana Veterinary College, 1923
Vouchers: Frank Hare and J. F. DeVine.

LAUGHLIN, NEWTON C.
1520 Parker Drive, Mayfield Heights, South Euclid, Ohio
V. S., B. V. Sc., Ontario Veterinary College, 1936
Vouchers: A. L. Miller and H. E. Myers.

MCCORMICK, HUGH C. 821 Wealthy St. S. E., Grand Rapids, Mich.
D. V. M., Ohio State University, 1935
Vouchers: E. C. W. Schubel and Walter K. York.

McQUISTION, RAY B. Trout Creek, Mich.
D. V. M., Grand Rapids Veterinary College, 1917
Vouchers: E. C. W. Schubel and F. K. Hansen.

MYERS, PHILIP 809 Flores Ave., Laredo, Texas
B. S., Texas A. & M. College, 1925
D. V. M., Texas A. & M. College, 1936
Vouchers: U. E. Marney and Chas. W. Neal.

NAVARRO, MANUEL 527 University Ave., Saint Paul, Minn.
D. V. M., Colorado State College, 1933
Vouchers: Joab P. Foster and John S. Dick, Jr.

NELMS, EMERY E. Smithfield, N. C.
D. V. M., Chicago Veterinary College, 1916
Vouchers: A. A. Husman and L. J. Faulhaber.

ROCHE, JOHNSON R. Gowrie, Iowa
D. V. M., Iowa State College, 1923
Vouchers: J. D. Thrower and T. W. Munce.

SMITH, WILLIAM R. Box 1365, Las Vegas, Nev.
D. V. M., Ohio State University, 1912
Vouchers: Edward Records and L. R. Vawter.

SPRAGUE, ARTHUR W. 519 Live Stock Exchange Bldg., Omaha, Neb.
D. V. M., Iowa State College, 1910
Vouchers: Paul L. Matthews and W. T. Spencer.

Applications Pending

SECOND LISTING

(See May, 1937, JOURNAL)

Bond, Eric W., Central Experimental Farm, Ottawa, Ont.
Cowen, Russell L., 452 Federal Bldg., Salt Lake City, Utah.
Lambert, Reuben C., c/o Montrose Beef Co., 118 N. Main St., Pittston, Pa.
Malaga Alba, Aurelio, Av. Costanera 105 Miraflores, Lima, Peru.
McComb, James A., 375 South St., Jamaica Plain, Boston, Mass.
Phifer, Lt. William G., CCC Co. 4407, Haughton, La.
Ramsey, Edward W., Brady, Texas.
Roth, Leon, 258 West 19th St., New York, N. Y.
Whitehead, Jackson O., 3312 Leeland Ave., Houston, Texas.
Williams, Harry U., Yerington, Nev.

The amount which should accompany an application filed this month is $7.91, which covers membership fee and dues to January 1, 1938, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. June 7-8, 1937. Dr. F. Y. S. Moore, Secretary, McAlester, Okla.

San Diego County Veterinary Medical Association. San Diego, Calif. June 8, 1937. Dr. Donald E. Stover, Secretary, Zoölogical Research Bldg., Balboa Park, San Diego, Calif.

Texas, State Veterinary Medical Association of, and A. & M. College of Texas Short Course for Veterinarians. College Station, Texas. June 8-10, 1937. Dr. M. B. Starnes, Corresponding Secretary, Public Health Dept., Dallas, Texas.

Kansas City Veterinary Association. Auditorium, General Hospital No. 1 Annex, Kansas City, Mo. June 15, 1937. Dr. C. W. Schulz, Secretary, 800 Woodswether Rd., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. June 16, 1937. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Ohio State University Veterinary Conference. Columbus, Ohio. June 16-18, 1937. Dr. Oscar V. Brumley, Dean, Ohio State University, Columbus, Ohio.

California State Veterinary Medical Association. Long Beach, Calif., June 21-23, 1937. Dr. Chas. J. Parshall, Secretary, Brentwood, Calif.

American Association for the Advancement of Science. Denver, Calif. June 21-23, 1937. Dr. Chas. J. Parshall, Secretary, Smithsonian Institution Bldg., Washington, D. C.

Eastern Iowa Veterinary Association. Mechanicsville, Iowa. June 22, 1937. Dr. John J. Strandberg, Secretary, 1005 Eighth Ave., Belle Plaine, Iowa.

Michigan State Veterinary Medical Association. Michigan State College, East Lansing, Mich. June 22-23, 1937. Dr. E. C. W. Schubel, Secretary, Blissfield, Mich.

Missouri Veterinary Medical Association. Hotel Ruff, Marshall, Mo. June 24-25, 1937. Dr. C. L. Campbell, Secretary, 1817 Holmes St., Kansas City, Mo.

North Dakota Veterinary Medical Association. North Dakota Agricultural College, Fargo, N. Dak. June 24-25, 1937. Dr. Lee M. Roderick, Secretary, State College Station, Fargo, N. Dak.

Wisconsin Veterinary Medical Association. Madison, Wis. June 29-30, 1937. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Houston Veterinary Association. Houston, Texas. July 1, 1937. Dr. Claude Canion, Secretary, 409 Link Rd., Houston, Texas.

Minnesota State Veterinary Medical Society and University of Minnesota Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 1-2, 1937. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. July 6, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. July 7, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

New York State Veterinary Medical Society. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. July 7-9, 1937. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Georgia State Veterinary Medical Association. Columbus, Ga. July 8-9, 1937. Dr. J. E. Severin, Secretary, 357 Edgewood Ave., S. E., Atlanta, Ga.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. July 12, 1937. Dr. W. H. Riser, Secretary, Glenwood, Iowa.

Willamette Valley Veterinary Medical Association. (Joint meeting with Northwest Veterinary Medical Association.) Multnomah Hotel, Portland, Ore. July 12-14, 1937. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Northwest Veterinary Medical Association. (Joint meeting of the Oregon, Washington and British Columbia Veterinary Medical associations.) Multnomah Hotel, Portland, Ore. July 12-14, 1937. Dr. B. T. Simms, Secretary, Oregon State College, Corvallis, Ore.

South Carolina Association of Veterinarians. Anderson, S. C. July 13-14, 1937. Dr. R. A. Mays, Secretary, 408-410 State Office Bldg., Columbia, S. C.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. July 14, 1937. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 14-15, 1937. Dr. Frank Hare, Secretary, 154 Walnut St., Lexington, Ky.

North Carolina State Veterinary Medical Association. (Joint meeting with Virginia State Veterinary Medical Association.) Wrightsville Beach, N. C. July 15-16, 1937. Dr. J. H. Brown, Secretary, Tarboro, N. C.

Virginia State Veterinary Medical Association. (Joint meeting with North Carolina State Veterinary Medical Association.) Wrightsville Beach, N. C. July 15-16, 1937. Dr. A. J. Sipos, Secretary, 1102 State Office Bldg., Richmond, Va.

Montana Veterinary Medical Association. Great Falls, Mont. July 22-23, 1937. Dr. Hadleigh Marsh, Secretary, Agricultural Experiment Station, Bozeman, Mont.

Western Michigan Veterinary Medical Association. Ionia Fair Grounds, Ionia, Mich. July 29, 1937. Dr. Chas. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

Western New York Veterinary Medical Association. Dr. F. L. Stein's farm, West Webster, N. Y. July 29, 1937. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

American Veterinary Medical Association. Hotel Fontenelle, Omaha, Neb. August 16-20, 1937. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

STATE BOARD EXAMINATIONS

Iowa Veterinary Medical Examining Board. State Capitol, Des Moines, Iowa. June 15-16, 1937. Applications must be on file at the Division of Animal Industry not later than 8:00 a. m., on June 15. Further information may be obtained from Dr. H. A. Seidell, Secretary, State House, Des Moines, Iowa.

California Board of Examiners in Veterinary Medicine. Long Beach, Calif. June 21-23, 1937. Applications must be filed with the Secretary at least 30 days prior to date of examination. Dr. Nelson E. Clemens, Secretary, 183 Castro St., Hayward, Calif.

Massachusetts Board of Registration in Veterinary Medicine. Department of Civil Service and Registration, State House, Boston, Mass. June 30-July 1, 1937. Applications may be obtained from the Secretary. Dr. E. W. Babson, Secretary, Gloucester, Mass.

Connecticut State Board of Veterinary Registration and Examination. State Office Building, Hartford, Conn. July 6, 1937, at 9:00 a. m., D. S. T. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

Virginia State Board of Veterinary Examiners. Murphy's Hotel, Richmond, Va. July 14, 1937, at 8:30 a. m. This will be the annual meeting and examination for license to practice in the state. Dr. H. H. Adair, Secretary, Bristol, Va.

Illinois State Board of Veterinary Examiners. Chicago, Ill. July 19-20, 1937. Dr. L. A. Merillat, president of the Examining Committee, asks candidates to file their applications with the Directors of Registration and Education, Springfield, Ill., not later than July 1.

ANAPLASMOSIS IN CATTLE*

By H. SCHMIDT, *College Station, Texas*
Texas Agricultural Experiment Station

Smith and Kilborne,[1] in the course of their classical work on Texas or southern fever, observed and described small, coccus-like bodies located peripherally in the red blood-cells in animals suffering from Texas fever and considered them as a developmental stage of the parasite which they found in the blood corpuscles of such cattle, namely, *Piroplasma bigeminum,* and which parasite they considered as the true Texas fever parasite. Today we know that they saw two different parasites and that the Texas fever they studied was a mixed infection. This fact was first recognized by Theiler,[2] in South Africa, in 1910, when he encountered pure infections with small coccus-like bodies in the red blood-cells and recognized them as the cause of a separate and distinct disease, anaplasmosis.

Based upon his observations, Theiler believed these bodies to be a protozoan parasite and established the genus *Anaplasma,* so named because no protoplasa appeared in perfectly stained specimens. He observed that in some cases these bodies were located on the margin of the red blood-corpuscles and later found cases in which the parasite was located more in the center of the red blood-corpuscles. Since a difference in virulence seemed to exist between the two types, Theiler set up two species, namely, *A. marginale* and *A. centrale,* the latter species being the less virulent of the two. After Theiler published his observations, the disease has been recognized in many parts of the world independently of piroplasmosis.

Theiler's view of the protozoan nature of *Anaplasma* did not remain unchallenged and a number of arguments have been presented for and against the view. These views will not be discussed here but for our purpose the appearance of the "peripheral bodies" (*Anaplasma marginale*) in the red blood-cells of a sick animal is acceptable proof that the animal is suffering from anaplasmosis.

It will not be possible to give a thorough review of all available literature on the subject of anaplasmosis in a paper of reasonable length to be presented here. May I, therefore, be pardoned if some phases of the disease, especially such phases as should be found in every text-book on veterinary medicine, are touched upon only lightly. Especially shall I pass over the car-

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

rier question, since that phase of the disease will be presented in another section of this meeting.

Anaplasmosis of cattle is an infectious disease manifesting itself in fever, anemia as a result of a rapid destruction of the red blood-corpuscles and degeneration of the large parenchymatous organs. These three cardinal manifestations are responsible for a series of other symptoms that can be observed in varying degrees of intensity in the affected animal and depending upon the degree of multiplication attained by the anaplasma; such are pallor of the mucous membranes, thin, watery blood, frequently a yellow discoloration of the skin and mucosae, depressed appetite, gauntness, weakness, constipation with hard, dry feces, a weak, rapid pulse, rapid respiration, drolling of saliva, and, when recovery is taking place, a marked craving to eat dirt. The urine may be highly colored but shows no tinge of red. The intensity of these symptoms varies markedly in different cases depending entirely upon the degree of multiplication attained by the parasite and thus the number of red blood-cells destroyed, the age of the animal affected and the season of the year. Thus, young animals up to about a year old may show only a rise in body temperature which is only detected by the systematic use of the clinical thermometer. Such cases are never observed by the practicing veterinarian because the owner himself would never recognize anything amiss with the animal. Young animals are remarkably resistant to anaplasmosis. The older the affected animal and the higher the atmospheric temperature, the more marked and numerous will become the symptoms shown by the animal. In adult animals a heavy mortality may be expected, especially if they become infected in summer. Thus, if one wishes to make a distinction in the severity of symptoms exhibited, one may speak of mild or severe, of peracute, acute and chronic cases or one may choose any other designation that strikes one's fancy.

At autopsy, one observes a more or less emaciated carcass showing marked anemia and icterus. The blood is watery, the heart is flabby and shows petechiae. There may be an accumulation of fluid in the pleural and pericardial sacs. The lymph-nodes are edematous, the lungs edematous and yellowish, the liver enlarged and on section brownish-yellow in color. The gall-bladder is distended with a thick, grumous, dark green bile. The spleen is enlarged and on section a dark red, almost black, soft pulp bulges out. Intestinal catarrh is usually present.

In nature, anaplasmosis is known to be transmitted by ticks. Thus, Theiler definitely established that it is transmitted by

Boöphilus decoloratus and *Rhipicephalus simus*. In Argentina, *Boöphilus microphilus*, and in Brazil, *B. australis;* in the United States, *Boöphilus bovis* and *Dermacentor occidentalis* transmit the disease. Zeller and Helm,[3] in Germany, showed that *Ixodes ricinus* also can transmit the disease. In all these cases the disease is transmitted by larvae obtained from ticks that had fed on infected animals. Other ticks that transmit the disease are *Rhipicephalus sanguineus, Dermacentor andersoni, D. albipictus,* and *D. variabilis,* which takes up the infection as a larva and transmits it to the following nymphal stage. The latter tick may also take up the infection as a nymph and transmit it in the following adult stage, which type of transmission it shares with *Rhipicephalus bursa*. In the case of *Hyalomma lusitanicum*, Sergent, Donatien, Parrot and Lestoquard[4] believe that the infection is taken up as the adult and transmitted by the adult in the next generation.

Numerous attempts have been made to transmit anaplasmosis by means of arthropods other than ticks, but thus far Sanborn[5] and his co-workers are the only ones who report the transmission of a single case. Artificially, however, a number of cases are on record in which the disease was transmitted from carrier animals to healthy animals by means of dehorning clippers, the hypodermic needle or even the bleeding trocar. To those cases already recorded, the author wishes to add two more outbreaks in Texas from which he has recently had an opportunity to examine blood by inoculation tests, one of which was spread in the herd by means of dehorning clippers and the other by means of the hypodermic needle. In neither case were antiseptic precautions practiced. In both instances heavy losses were sustained and both premises had been free from Texas fever-carrying ticks for a number of years. In three other recent outbreaks in the same state and diagnosed by the author as anaplasmosis, either by blood inoculation or by autopsy, no special transmitting agency could be incriminated.

Susceptibility, Immunity and Immunization

All cattle are susceptible to anaplasmosis but there is a wide difference in the severity of the disease depending mainly upon the age of the animal. In animals up to about one year old, the reaction to infection may be so mild that it escapes the attention of the trained observer. The atmospheric temperature also has a marked influence upon the severity of the reaction insofar as losses from infections occurring during the warm season of

the year are much heavier than during the cooler season. In the summer the mortality in adult cattle may run to 50 per cent or over. These statements also hold true for piroplasmosis.

Animals recovering from an attack of anaplasmosis remain carriers of the infection for a long time, probably for life. Offspring from such carriers are as susceptible to infection as offspring from susceptible parents.

Based upon these facts, Dr. Mark Francis, at the Texas Agricultural Experiment Station, and Dr. J. W. Connaway, at the University of Missouri, about 1895, worked out a method of immunization of young animals to so-called Texas fever, and Francis practiced it extensively in introducing pure-bred cattle into Texas. The author took over this work in 1913 and has since immunized against Texas fever hundreds of animals introduced into Texas and other countries to the south. During this routine immunization it was recognized that our Texas fever is a mixed infection of piroplasmosis and anaplasmosis and much information was collected regarding the character of anaplasmosis in Texas.

A careful record was kept of all inoculations, including the source of blood, amount injected, day of injection, and the daily body temperature for 55 to 60 days following the injection. All temperatures were taken beginning at 7:00 a. m. and usually were completed by 8:00 a. m., depending upon the number of animals under observation. Inoculations were practiced between November 1 and March 1, in rare exceptions later in March. In most cases the virulent blood was drawn from a tick-infested animal; in some cases from an animal that had been free of ticks for from one to five years or had never had ticks on it but had been inoculated at least one year previously with blood containing both anaplasmata and piroplasmata. The animals immunized all came from tick-free territory and from many states of the Union.

On account of the widespread prevalence of anaplasmosis as well as piroplasmosis in the vast area of Texas then infested by ticks, it was, of course, necessary to immunize such cattle against both piroplasmosis and anaplasmosis. A splendid opportunity was afforded, therefore, to study many cases of the two diseases where one would follow upon the heels of the other in the same animal. The data recorded are so extensive that no attempt will be made here to give many individual records in detail, but instead the animals will be discussed in comparable groups wherever they have been inoculated with blood from the same animal. It was

not possible, of course, to follow all reactions to the inoculations with the microscope in a study of blood-smears. This was usually done in some animals out of each group to make sure that the fever reaction following the inoculations was really that of piroplasmosis or anaplasmosis. Thus, when the animal was inoculated with blood containing both piroplasmata and anaplasmata, two distinct fever reactions would follow. The first reaction, due to piroplasmata, would begin anywhere from the third to the 16th day and the second reaction, due to anaplasmata, would begin anywhere between the 17th and 48th days. The longer period of incubation, however, is exceptional. Thus, of 309 animals inoculated from two different blood-donors, and checked on this point only, nine had a period of incubation of 45 to 48 days; in 187 animals the period of incubation ranged from 29 to 38 days, and 17 had a period of incubation of 17 days. In 19 two-year-old bulls inoculated on the same day, each with 1.5 cc of fresh defibrinated blood drawn from the same animal and harboring only anaplasmata, the period of incubation, as indicated by the body temperatures and checked by microscopical examination in seven of them, ranged from 29 to 38 days, as is indicated in table I.

TABLE I—*Animals reacting in a group of* 19 *two-year-olds.*

PERIOD OF INCUBATION (DAYS)	29	30	31	32	33	34	35	36	37	38
Animal reacting	2	0	6	0	3	1	5	1	0	1

We are here concerned only with anaplasmosis but since the upper extreme of the period of incubation of piroplasmosis and the lower extreme of the period of incubation of anaplasmosis almost coincide and the fever reactions may thus overlap, it became necessary to keep this in mind to make sure that the animal had reacted to both organisms and would thus develop an immunity to both.

The behavior of the body temperature during the fever reaction varies considerably from animal to animal, both as concerns the duration and height of the fever. Thus, the duration may range from one to two days, to two weeks or over, of continuous fever or the fever may be interrupted by days on which the temperature is normal. The fever may rise gradually or abruptly and may terminate in the same way. The chart in figure I gives the temperature curves for two animals suffering

from anaplasmosis. These bulls were two years old and were inoculated at the same time, with the same amount of blood drawn from the same animal. The fever curve of bull 3 is rather extremely atypical but clearly illustrates the interruption of days of fever by days of normal temperature. Bull 11 shows a rather typical curve for a more severe case both as regards height and duration of fever. Bull 2 (fig. 2) is part of the same group as bulls 3 and 11 and was inoculated with them and in the same way. His death was rather unexpected. He walked from the corral into his stall for his evening grain feed, took a mouthful and dropped dead.

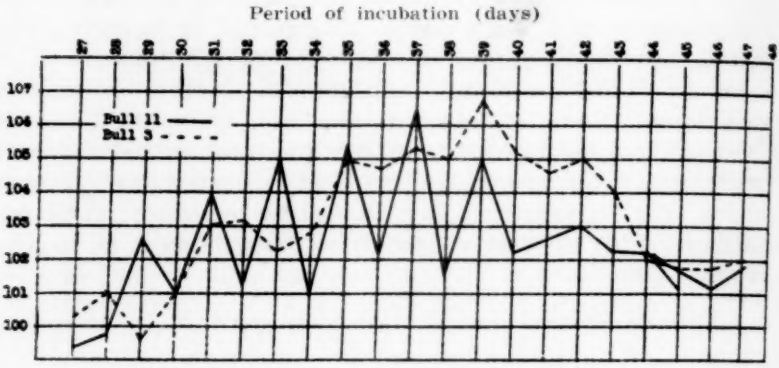


FIG. 1. Temperature curves of bulls 11 and 3.

The temperature curve of animal 139, as shown in the chart in figure 2, is not at all unusual for young animals. In the course of the 59 days that its temperature was taken daily following inoculation, it showed a rise above normal on the 21st, 27th, 31st and 33rd days, but anaplasmata could not be found in its blood. Since this animal had been inoculated from a suspected field case of anaplasmosis found in a region where anaplasmosis was not known to exist, it was rather important to get a clear-cut diagnosis. For that reason the blood serum of animal 139 was subjected to the serum-distilled water test and also an aged susceptible cow was inoculated with 8 cc of fresh whole blood drawn from it. The serum-distilled water test was negative 24 days after the last rise in body temperature, while the cow inoculated with the blood showed a typical fever curve with many anaplasmata in the blood. Probably the negative result of the serum-distilled water test should have been expected as a result of the mild fever shown by the animal.

Usually the animal shows no symptoms of illness until the temperature reaches 105° F. When this temperature is reached, the animal, if it shows anything at all, reacts with depression and loss of appetite. If, thereafter, the body temperature continues high, the other symptoms as already enumerated will become manifest. We have never observed hemoglobinuria. The mortality from anaplasmosis among more than a thousand animals immunized at the age of 15 months or under was 1.3 per cent. In older animals, however, heavier losses may be expected. Thus, of 19 two-year-old bulls inoculated with a pure strain of anaplasma, four died from anaplasmosis.

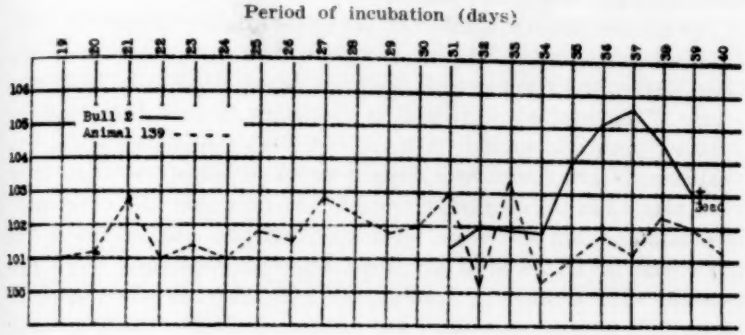


FIG. 2. Temperature curves of bull 2 and animal 139.

As the animals recover from the disease, a marked desire to eat dirt is usually observed for a week or longer.

The anaplasma rapidly destroys the red blood-cells of the animal during the fever period, so that in addition to the high fever one observes a thin watery blood. The red-cell count may sink to below 2,000,000. At this stage the other symptoms such as loss of appetite, depression, fast, weak pulse, rapid respiration, dribbling saliva, jaundice, weakness and rapid loss of condition become manifest.

A microscopical examination of the blood during the fever period will reveal many red cells when stained with Giemsa's stain to contain bluish-red bodies located near the margin of the cell or sometimes nearer the center. These vary in size from small specks to about 0.9 micron in diameter, are more or less round but sometimes are distinctly angular. The infected cells contain from one to three such bodies, rarely four. A case of *A. centrale*, as described by Theiler and others and in which the organisms appear only in the center of the red blood-cells, has

not been encountered by the author in Texas. In the majority of cases these bodies are already present in the blood at the time the fever appears and as the fever continues and rises the number of infected red cells increases until as many as 50 per cent may appear infected. As recovery takes place, the number of infected red cells decreases and by the time the body temperature is back to normal the bodies can no longer be found. Before this stage is reached, however, other changes are found in the blood, especially if the temperature remained around 104 or 105° F. or over for several days. These changes are the result of blood regeneration and consist in anisocytosis, polychromasia, basophilia and sometimes nucleated red cells.

The author has frequently run into cases of anaplasmosis artificially transmitted in which the organisms did not appear in the blood during the fever period. In such cases, especially if it was a case of diagnostic injection, it is necessary to inoculate another known susceptible animal from the one first inoculated until the anaplasmata are found or sufficient proof is obtained that they were present in the original material. The fact that the originally inoculated animal shows no fever reaction and anaplasmata are not found in its blood is no proof that anaplasmata were not present. The chances of finding the anaplasmata when mature susceptible animals are used for inoculation are much greater than when yearlings are used. As an additional test the serum-distilled water test may also be applied but this test needs further study before it can be accepted as infallible. In this test a drop of clear serum from the animal to be tested, when added to about 1 cc of distilled water, produces a readily recognizable turbidity. The inability to find the organism in a test animal may become embarrassing as it has to the author, especially if it should be a question of diagnosis of a suspected outbreak of anaplasmosis in a part of the country where anaplasmosis has not been suspected and a quick diagnosis is very desirable. The inoculation test is, of course, only of interest in cases where the animals suspected of suffering or having died of anaplasmosis have never been infected with Texas fever ticks.

An interesting question is that of the length of time the anaplasmata can live in blood drawn from the animal. This is important especially if animals are to be immunized against Texas fever, which is here considered as a mixed infection of piroplasmata and anaplasmata. Mohler,[6] in Farmers' Bulletin 569, makes the statement "that such blood may be kept in a dark, cool place for one or more months without deteriorating and may be

shipped to other points for use." Since it is a question of using such blood for immunizing animals, the inference is that the organisms are still alive at the end of that time.

It is also important in determining the results of attempted cultures of anaplasmata. Thus, Helm,[7] reports multiplication of anaplasmata in his cultures up to 37 days. He kept his cultures at room temperature. Veglia,[8] reports that in his cultures, regardless of whether they were kept at 7° C., 15° C., or 26° C., multiplication of anaplasmata continued in one case up to 40 days but more frequently for 21 to 26 days. He used various media among which infected blood is included. The author has drawn blood from carrier animals and kept it at room temperature or at ice-box temperature for varying lengths of time and then injected 7 cc subcutaneously into susceptible animals to determine whether the piroplasmata or anaplasmata were still living.

TABLE II—*Showing the survival period of Anaplasma marginale and Piroplasma bigeminum in infected blood kept at room temperature.*

Cow	Days Blood Kept at Room Temperature	Amount of Defibrinated Blood Injected (cc)	Results
104	26	7.0	Negative
101	13	7.0	Negative
102	11	7.0	Negative
117	11	7.0	Negative
105	10	7.0	Negative
115	10	7.0	Negative
105	9	7.0	Negative
113	9	7.0	Anaplasmata
102	8	7.0	Anaplasmata
106	8	7.0	Anaplasmata
112	7	7.0	Anaplasmata
111	6	7.0	Anaplasmata Piroplasmata (?)
111	5	7.0	Piroplasmata (?)

Note: The question mark indicates that piroplasmata could not be found but that the type of fever reaction indicated their presence.

It was found that at room temperature *P. bigeminum* would live for four days and probably for six days, but not for seven days, while *A. marginale* would live for nine days but not for ten days. At ice-box temperature *P. bigeminum* would live for ten days and probably for eleven days, but not for twelve days, while *A. marginale* lived for eleven days but not for twelve days.

TABLE III—*Showing the survival period of Anaplasma marginale and Piroplasma bigeminum in infected blood kept at ice-box temperature.*

Cow	Days Blood Kept at Ice-Box Temperature	Amount of Defibrinated Blood Injected (cc)	Results
107	7	7.0	Anaplasmata and piroplasmata
108	8	7.0	Anaplasmata and piroplasmata
109	9	7.0	Anaplasmata and piroplasmata
112	9	7.0	Piroplasmata (?)
114	9	7.0	Anaplasmata and piroplasmata
110	10	7.0	Anaplasmata, piroplasmata (?)
115	10	7.0	No anaplasmata, no piroplasmata
116	10	7.0	Piroplasmata
116	11	7.0	Anaplasmata, piroplasmata (?)
118	12	7.0	No anaplasmata, no piroplasmata

Note: The question mark indicates that piroplasmata could not be found but that the type of fever reaction indicated their presence.

In the author's culture experiments in which 1 cc of defibrinated blood drawn from an animal showing a heavy infection of the red blood-cells with anaplasmata was added 5 cc of fresh defibrinated blood drawn from a susceptible animal and to this mixture 0.1 cc of 50 per cent sterile glucose solution was now added, an increase in red blood-cells infected with anaplasmata did not occur when kept at room temperature. Furthermore, the inoculation of such cultures into susceptible cattle after 16 days did not result in anaplasmosis of such cattle. Since 1 cc of blood from a carrier animal injected into a susceptible animal almost invariably produces anaplasmosis, the conclusion is justified that the anaplasmata had already died out in the cultures. This is borne out by the results obtained in the experiments in which the survival period of anaplasmata in infected blood was determined. It thus seems that the bodies that Helm and Veglia saw in their cultures were either not anaplasmata or the organism had changed to a non-virulent form.

TREATMENT

In recent years a number of new drugs have been recommended for the treatment of anaplasmosis but as rapidly as one writer advances claims of success with a certain drug another writer publishes an unfavorable one. At the present time, there is no drug known that gives satisfactory results in the treatment of anaplasmosis. In our immunization work the writer has al-

ways relied upon symptomatic treatment in those animals demanding medical care. In this a laxative feed is considered of paramount importance. It forestalls an early appearance of constipation and thus tends to insure the proper elimination of toxic products from the bowel. For this purpose wheat bran constitutes one-half of the concentrate feed and for roughage only a good quality of pea green alfalfa hay is fed. Green grazing, especially on young green grain fields, is very desirable.

Further treatment depends entirely upon the body temperature and the appetite of the animal. As long as the temperature remains below 105° F., a loss of appetite is given no consideration and seldom occurs. If the temperature rises above 105° F. and the animal shows depression, a large dose of Epsom salt in hot water is given. This will physic the animal in from eight to ten hours and usually depresses the temperature by the end of 24 hours by one degree or more and whets the appetite. Should the temperature fail to decline, or if it should even rise and the weather be warm, then the animal is placed in a cool place and wetted down with cool running water for periods of 15 minutes with half-hour intervals to control the temperature. Rectal injections of cold water may also be practiced. Every precaution is taken to avoid over-exertion of the animal; strangers and other animals are not allowed to come near it.

Occasionally an animal is found which, after having withstood a high body temperature, will continue to refuse all feed even after the body temperature has returned to normal for some days. Continued use of drugs that stimulate the rumen such as nux vomica, barium chloride, tartar emetic or nicotine sulfate usually will have the desired effect, especially if the animal is tempted with fresh green grazing. Sometimes such animals may remain lying down for ten days or so, although able to get up, but as a rule they are so weak that they remain standing only for a half-minute or only long enough to change resting places.

After the animal has recovered from anaplasmosis, a serious problem remains, namely, to determine what to do with it. This question cannot be answered intelligently until the carrier problem is solved and the present distribution of the disease determined. As far as the area still infested with the Texas fever tick or released areas in which animals are still living that once were infested with this tick are concerned, they may be safely considered as infected territory. But there are also large areas of released territory that have been free of the disease for a long time and also vast areas of territory in which the Texas

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fever tick had never gained a permanent foothold in which the disease is now found. As long as recovered animals are permitted to live in such territory, they constitute a real and constant danger for the remaining animals, and any one of these areas is liable to be the starting point of a severe outbreak of the disease at any time.

It may, therefore, be well worth while for the proper agencies to determine whether it would not be feasible to dispose of such recovered animals and thus remove this constant source of danger. There are, of course, a number of difficulties to be overcome, the greatest of which is to find the original carriers from which the outbreak originated. Such animals could be discovered only by the inoculation of test animals under the presumption that the carrier state is of long standing. In the case of recently recovered animals, the serum-distilled water test may constitute a valuable aid, but over how long a period after recovery it will give positive results is not definitely known. The writer has run some tests on a limited number of animals to ascertain this point and has found the test negative eight months after recovery from the disease. No test was run earlier than eight months after recovery. In many cases the owner himself, once he has experienced losses from the disease and knows the danger to which his herd is constantly exposed when carrier animals are present in it, is very anxious to find such and dispose of them.

Another alternative is to immunize all young animals each year. This the writer cannot recommend at this time, because it would make carrier animals out of all of them and create a constant menace for susceptible animals on adjoining premises and eventually give the disease a permanent foothold in this country.

REFERENCES

[1]Smith, T., and Kilborne, F. L.: Investigations into the nature, causation, and prevention of Texas or southern cattle fever. U. S. Dept. Agr., B. A. I. Bul. 1 (1893).

[2]Theiler, A.: *Anaplasma marginale* (Gen. and Spec. nov.). The marginal points in the blood of cattle suffering from a specific disease. Rpt. gov. vet. bacteriologist, Transvaal (1908-1909), pp. 7-64.

[3]Zeller, H., and Helm, R.: Versuche zur Frage der Übertragbarkeit des Texas-fiebers auf deutsche Rinder durch di bei uns vorkommenden Zecken Ixodes ricinus und Haemaphysalis punctata cinnabarina. Berl. Tierärzt. Wchnschr., xxxix (1923), pp. 1-4.

[4]Sergent, E., Donatien, A., Parrot, L., and Lestoquard, F.: Tiques et piroplasmosis bovines d'Algerie. Bul. Soc. Path. Exot., xxi (1928), pp. 846-849.

[5]Sanborn, C. E., Stiles, G. W., Moe, L. H., and Orr, H. W.: Transmission of anaplasmosis by flies. (Reprinted, Okla. Agr. Exp. Sta. Rpt. for 1930.)

[6]Mohler, J. R.: Texas or tick fever. U. S. Dept. Agr. Farmers' Bul. 569 (1914).

[7]Helm, R.: Beitrag zum Anaplasmen-Problem. Zeit. f. Infekt. d. Haust., xxv (1922), pp. 199-226.

[8]Veglia, F.: The cultivation of *Anaplasma marginale* in vitro. 3rd and 4th Rpts., Dept. Agr., Union So. Afr. (1915).

DISCUSSION

DR. W. H. BOYNTON: I shall give a little of our experience. We find that glucose solution, that is in heavy quantities, and sodium cacodylate apparently help the animal a great deal in this disease, providing you can get the animal soon enough. We have been using a 5 per cent glucose solution in distilled water and adding two grams of sodium cacodylate per hundredweight of the animal to be treated. We give a liter of this material intravenously. We had some very satisfactory results in bringing abou' prompt recovery and also helping the dairy animal come back on the milk. One of the symptoms of a dairy animal is going off milk very suddenly. By this treatment we have been able to bring back milking in a week or two.

DR. C. C. HASTINGS: When these southwestern cattle are shipped into the Corn Belt of the Central West and are infested with this parasite, is there danger of our native cattle getting it from them?

DR. SCHMIDT: Anaplasmosis is transmitted from animal to animal by some carrier. I enumerated a number of them in the paper. Up to now, ticks have been definitely incriminated, and Sanborn and his co-workers in Oklahoma think they have one authentic transmission by flies. We have had a number of outbreaks in Texas. We could not incriminate any carrier, but this may be due to the fact that if ticks are the carriers, the disease does not become manifest until about 100 to 120 days after infection has taken place. When you find the animal sick 120 days after the carrier has been there, you will have a hard time finding it. We do not think there is any danger of transmitting it in the Corn Belt because I do not believe you have the carrier in the Corn Belt. It is true that it was found in southern Kansas. I think it is found all through there. The carrier was not determined at the time.

DR. G. DIKMANS: In connection with this particular question that was raised here, I want to call attention to a few cases that occurred in Delaware. Although it was never traced, presumably it was from some carrier. Later on, there was an inoculation for anthrax, and from all that we were able to discover, the animal that came down with the disease came down with it as a result of that inoculation, because people were not thinking about it.

About a year or two ago, we learned of some cases in Mississippi. I tried my very best to find out where they came from. I endeavored to discover how they originated at that particular time of the year (it was in February), because, in a general way, we feel it is a disease of the summer, as Dr. Schmidt has said, especially when the temperature gets very high. At that time, those cows were tested for Johne's disease. We had that in that particular case. So I think that is one thing that should be guarded against. I think the Bureau has already issued instructions, especially to men engaged in Bang's disease eradication, urging the use of a disinfectant with a clean needle wherever it is possible, especially where the disease is present.

DR. HASTINGS: The reason I asked that question is because we have a large number of southwestern cattle shipped into the Corn Belt, and I have seen a number of those herds infected with this disease, but I have never seen it spread to native cattle, although I have seen these southwestern cattle die on the farms. They are usually shipped in in the fall of the year and they start to die in the winter, which bears out what Dr. Schmidt said about the 100-day period of incubation.

DR. T. H. FERGUSON: I should like to ask if this disease occurs in swine.

DR. SCHMIDT: There is an anaplasma that has been reported in hogs, but we have not got it. Doyle, I think, reports some anaplasma-

like bodies in hogs from Indiana. If you will go back to the JOURNAL of the American Veterinary Medical Association, you will find his report* there. I do not think they were anaplasma. An anaplasmosis occurs also in sheep, but in these animals the disease is probably never recognized because anaplasma apparently are harmless bugs there. I have tried a number of times to transmit bovine anaplasma through sheep but I did not get the results that other workers have reported. Mine were all negative.

DR. L. A. MERILLAT: Dr. Schmidt, you probably recall that Dr. DuToit, of South Africa, at the Twelfth International Veterinary Congress referred to immunizing cattle destined for South Africa from continental Europe by a simple injection of *Anaplasma centrale*. Is that a practice that is developed, or is it done here, or is there any merit to it? It seemed to be a controversial question at the time.

DR. SCHMIDT: I brought that point out in my paper. I never found *Anaplasma centrale* in Texas. I do not know whether we have it in the United States; no one has reported it so far. They have *Anaplasma centrale* and *Anaplasma marginale* in South Africa. They use the immunity set up by the *A. centrale* against the *A. marginale*. DuToit injects the *A. centrale* first and later injects the *A. marginale*. That, however, is necessary only when you are trying to immunize an old animal. When you immunize an animal that is not more than 15 months old, you will hardly need to practice that precaution, as borne out by the results I reported.

Women's Auxiliary to the A. V. M. A.

The Women's Auxiliary to the A. V. M. A. is just completing the fifteenth year of handling a loan fund for senior students in veterinary colleges. During that time the Auxiliary has loaned a total of $11,500.00 to 48 students and has increased the fund from $530.00 to $4,930.00. Is your wife a member of this organization? Dues are $1.00 per year. Remittances should be sent to Mrs. S. E. Hershey, Secretary.

The complete personnel of officers of the Auxiliary for 1936-37 is as follows:

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When rogues fall out, honest men get into their own.—Sir Mathew Hale.

*Doyle, L. P.: A rickettsia-like or anaplasmosis-like disease in swine. Jour. A. V. M. A., lxxxi (1932), n.s. 34 (5), pp. 668-671.

HEMORRHAGIC SEPTICEMIA INVESTIGATIONS

The Significance of Pasteurella Boviseptica Encountered in the Blood of Some Florida Cattle*

By D. A. SANDERS, *Gainesville, Fla.*
Florida Agricultural Experiment Station

The Pasteurella group of organisms is known to have a widespread distribution. They have been reported occurring in soil, slow flowing and stagnant water, dead organic matter, digestive and respiratory tracts of wild and domestic animals and in the blood and tissues of laboratory animals that have succumbed to exposures of the organism. Pasteurella have been demonstrated in the tissues of animals which died from non-bacterial diseases, notably those of filtrable virus origin, including feline infectious enteritis,[1] canine distemper and hog cholera.[2] Moore[3] examined the upper respiratory passage of three healthy cattle and isolated organisms not distinguishable from Pasteurella. Jorgenson[4] studied the bacterial flora of the upper respiratory passage of 250 normal cattle. He concluded that *Pasteurella boviseptica* was an inhabitant of the upper respiratory tract of a considerable percentage of normal cattle and that cold and fatigue predispose animals to attacks of these organisms.

Scott and Farley[5] isolated *P. boviseptica* from the lungs of four general autopsy cases in cattle which had no visible lung lesions. These investigators report the presence of Pasteurella and other species of bacteria in the tissues secured from autopsies of cattle affected with shipping fever. Their studies suggest the causative factor of shipping fever to be exposure and improper feeding and care of animals, and that *P. boviseptica* might have a small place in the production of the final disease which caused death in exposed animals. Pasteurella are known to be the etiological agent in some cases of omphalophlebitis and on numerous occasions they have been reported as a primary and spontaneous blood-stream invader responsible for bovine hemorrhagic septicemia. The occurrence of hemorrhagic septicemia in herds of cattle has frequently been explained as a result of an increase in virulence of the organism and to predisponents which lower the body resistance. The disease in cattle is commonly diagnosed by means of clinical, postmortem and laboratory findings. The demonstration of Pasteurella in bovine carcass material, of suspicious hemorrhagic septicemia cases, and their virulence for

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

rabbits is often accepted as a criterion of the etiological significance of such organisms.

Samples of heart-blood and portions of internal organs of cattle, that died on the range and in dairy herds in various sections of Florida, have frequently been submitted to veterinary laboratories for diagnostic purposes. The usual history accompanying such material showed that the animals died in a sporadic manner and exhibited lesions suspicious of hemorrhagic septicemia. From this material, organisms were isolated which, upon a detailed study of their cultural, morphologic and biochemic characteristics and their pathogenesis for small test animals, showed they belonged to the Pasteurella group. They were identified as *P. boviseptica* and as a result studies were undertaken regarding possible etiological relationship of these organisms to the losses incurred. Owing to sporadic development of the cases within the herds, opportunity for making clinical or postmortem observations were limited. Realizing the different degrees of virulence attributed to strains of Pasteurella under variable conditions, no evaluation is claimed for organisms other than those encountered and examined in these studies or for their action on animals under conditions other than those undertaken in the investigation.

Materials

The work is being conducted in an isolation building, 25 x 16 feet, with reinforced concrete foundation and floors. The foundation extends ten inches beyond the outer walls of the building. A circumvallate moat six inches in width, built into this concrete extension and filled with strong disinfectant, precludes the entrance of crawling forms of life. The concrete is continued upward to a height of two feet above the floor level, forming part of the outer walls of the structure. Upon this two-foot concrete extension, timbers are bolted and the walls proper extend upward to a height of eight feet by means of screened-in framework. The building is covered by a hip roof having a three-foot eave.

Four pens, 6½ x 7½ feet, of steel-tube panel construction, set in a five-inch curb, are situated centrally within the building. Floors in the pens slope to a common drain six inches in width. Drainage is conveyed to a septic tank outside. Removable wooden slatted platforms of low construction are provided in the pens upon which the animals usually rest. An observation and exercise alley four feet in width surrounds the pens between the walls and panels. The building is entered through a vestibule, the

walls of which are of similar structure as those of the main unit. Water is supplied to each pen through pipes leading from the city main. The building is wired for electricity. A good grade commercial dairy ration and beet pulp is used as feed for the animals. The young calves receive milk from a pail.

Experimental Observations

Experiment 1: Attempts to produce clinical cases of bovine hemorrhagic septicemia were undertaken by exposing calves, yearlings and cows to Pasteurella which were isolated from the blood and tissues of cattle that died in the field. After isolation by rabbit inoculations, the organisms were grown and maintained on ferric salts agar or meat infusion broth containing ferric ammonium citrate.[5] Efforts made to isolate Pasteurella from the mucous membrane of the upper respiratory tract, the nasal secretions, and the blood of these animals before exposure resulted in negative findings. Methods of exposure undertaken consisted of feeding *P. boviseptica* in the form of 24-hour agar and bouillon cultures, by feeding the internal organs and peritoneal exudate of laboratory animals which died from acute septicemia following intraperitoneal injections or nasal insufflations of organisms, by spraying the nasal passages, conjunctival and aural cavities with saline suspensions of the organism and by introducing them into the sinuses of the head by means of a trephine.

Fourteen calves, ranging from one day to six months of age, were exposed to *P. boviseptica* by nasal spray and by ingestion. Amounts varying up to 50 cc of a 24-hour saline suspension were sprayed into the nasal passages and 800 cc of a 24-hour bouillon culture was given as a drench. Ten of the animals thus exposed exhibited no ill effects therefrom, while four which received a heavy nasal spray developed clinical symptoms of catarrh of the respiratory passage. In the four animals this condition was manifest by physical irregularities including increased rate of pulse and respirations, elevation of body temperature, inappetence, sneezing, mucopurulent nasal discharge and cough. In two of these cases the symptoms abated, the animals recovering their pristine condition in the course of a week. Two retained the cough for a period of eight weeks but remained in good physical condition.

Repeated attempts to recover Pasteurella from the blood-stream of these experimentally exposed animals were made during the clinical reactions. The neck was prepared by a thorough washing with soap and water. An area over the jugular vein was

selected, shaved and swabbed with 70 per cent alcohol, after which it was painted lightly with tincture of iodine. Blood in various amounts was collected directly into culture tubes and flasks containing Krack and Teasley's[6] blood-culture medium by use of a 16-gauge needle attached to and sterilized as an integral part of the bleeding apparatus. Whole blood was collected from the jugular vein with sterile glass syringes and transferred to ferric salts agar and meat infusion bouillon. All cultures were incubated at 37.5° C. for five days. Other blood samples were secured by means of sterile syringes, transferred to sterile tubes, and held at room temperature for observations. Under these conditions we have not been successful in recovering the organism from the blood-stream of cattle thus exposed to Pasteurella or in producing septicemia in any of its recognized forms. *P. boviseptica* was readily isolated by these methods from the blood-stream of clinically affected rabbits and birds which received intraperitoneal injections or nasal insufflations of the organisms.

P. boviseptica was readily recovered from the secretions of the nasopharynx of bovines thus exposed for a period of three months after introduction through the alimentary and nasal passages and from the frontal sinus two months after introduction by means of a trephine. These organisms, before exposure of the animals and after their recovery therefrom, were lethal for rabbits when 0.001 cc of a 24-hour ferric salts bouillon culture was injected into the peritoneal cavity or when a drop of the whole culture was instilled into the nasal passage. Autopsies of the calves which retained the cough following heavy experimental exposure by nasal spray was made after a period of eight weeks. The apical lobes of the lungs contained several pneumonic foci 0.5 cm in diameter. Pasteurella were recovered from the bronchioles adjacent to these lesions.

From the results obtained it was concluded that the index of resistance in bovines is normally effective against the invasiveness of mild exposures of Pasteurella, that the environment of the respiratory passage is favorable to the existence of these organisms and that in some cases of heavy experimental exposure by means of nasal spray Pasteurella gain the depths of the bronchioles which may result in development of pneumonic foci. It was also concluded that criteria other than simply bringing together the bacteria and the host animal were necessary for the organisms to be disseminated from the respiratory passage into the blood-stream.

Predisponents applicable to local conditions were thought to weaken the index of body resistance, thereby favoring the in-

vasive offenses of Pasteurella which would account for their presence in the blood. These phenomena, if present, would lead to penetration of the organism from its initial point of lodgment in the respiratory passage or other established foci, by contiguity into the sub-tissues and thence into the circulation, resulting in generalized invasion of the blood-stream. Attempts to lower the resistance of cattle exposed to nasal sprays of Pasteurella were undertaken by feeding virulent paratyphoid organisms and sublethal quantities of various stock-poisoning plants, by introducing sand into the alimentary canal and through anaphylactic shock. These factors failed to establish conditions necessary to bring about an invasion of the blood-stream by Pasteurella or the production of hemorrhagic septicemia in any of its recognized clinical forms.

Field Observations

In one dairy herd where losses of milk cows had been sustained, *P. boviseptica* was isolated from the jugular vein and heart-blood of an animal autopsied which had shown clinical evidence of disease only a short time. Upon examining prepared blood-smears of a clinically affected animal in the herd during this period, *Anaplasma marginale* Theiler was demonstrated parasitizing the erythrocytes. The simultaneous occurrence of *P. boviseptica* and *A. marginale* in the blood of animals of the same herd led to suspicion of a relationship between these agents which would explain the presence of Pasteurella organisms in the blood of cattle in many instances encountered in the field. This suspicion was further strengthened when, as shown above, Pasteurella organisms of bovine origin, with a high degree of invasiveness for small laboratory animals, failed in themselves to produce septicemia in cattle.

Considering the biological characteristics of *A. marginale*, its period of incubation, various types of infection produced, methods of reducing body resistance, and its widespread occurrence, it appeared reasonable to believe this hematozoön agent would serve as an associate, working in concert with Pasteurella, which would allow the organism to gain entrance to the blood-stream of bovines from their location in the respiratory passage or other established foci. Investigation regarding *A. marginale* serving as a predisponent in the production of conditions favorable to the invasive powers of *P. boviseptica* seemed warranted.

Experiment 2: This experiment was undertaken for the purpose of making observations on animals which were harboring *P. boviseptica* in the upper respiratory passage and at the same time approaching or passing through acute reactions of anaplas-

mosis. Two bull yearlings 14 months of age were exposed by nasal spray to *P. boviseptica*, using bouillon cultures and saline suspensions of 24-hour growth. Three strains of Pasteurella were used, namely F1, which was isolated from the carcass of a dead bovine; F2, isolated from the synovial fluid of a young calf suffering from septic joint infection, and B. S., isolated from the upper respiratory passage of a healthy cow. Before exposure, these organisms proved to be virulent for small laboratory animals. One one-thousandth cc of a 24-hour bouillon culture proved fatal for rabbits in 18 hours when injected into the peritoneal cavity or when a drop of the undiluted suspension was placed on the nasal mucosa. The yearlings withstood the exposure with no evident reaction. To determine if these animals were free from anaplasma infection or in a state of premunition, they were splenectomized. In general, the technique of de Kock and Quinland[7] was followed in performing the splenectomies. Recovery from the operation was uneventful in both animals. Healing of the operative areas by first intention was secured. Careful clinical observations and blood examinations made over a period of 30 days following removal of the spleen failed to reveal a relapse of anaplasmosis, indicating the animals had not previously been affected with the disease.

Thirty days following the splenectomy, all of these animals were intrajugular recipients of 5 cc of citrated blood obtained from a cow which was suffering from an acute attack of anaplasmosis. Acute reaction to the parasitic invasion of *A. marginale* occurred. Blood examinations and clinical symptoms during the attack showed a typical picture of anaplasmosis. Approximately 50 per cent of the erythrocytes were parasitized. These resulted marked anemia, anisocytosis, poikilocytosis and oligocythemia, with prostration and death on the 13th and 15th days following the anaplasma inoculations. Whole blood and blood cultures secured under aseptic conditions as previously described and blood smears from the jugular vein during the early stages of the acute attack of anaplasmosis on these yearlings failed to reveal the presence of Pasteurella.

Both animals died during the night and autopsies were held the following morning. The skin, visible muscosa and subcutaneous connective tissue presented a yellowish discoloration. The blood and the muscle tissue were pale. A marked flecking of the pericardium and epicardium with petechial hemorrhages occurred. Extensive subendocardial sugillations were present. The lungs presented areas of congestion and the mediastinal lymphglands were edematous. There were prominent subcortical petech-

ial hemorrhages of the kidneys. The liver was congested and the gall-bladder distended with dark, thick bile. Catarrhal enteritis and evidence of intestinal hemorrhage were present.

The hearts were removed by ligating and severing the large blood-vessels. Cultures of the heart-blood were made and incubated at 37.5° C. Samples of the heart-blood were collected in sterile tubes and held at room temperature for observation. After 24 hours, smears made from the whole blood and stained by Giemsa's or Wright's method showed organisms which, after being isolated in pure culture from the blood-samples and submitted to the salient tests, were found to be Pasteurella, similar to those originally introduced into the nasal passage.

Experiment 3: Having made observations on animals harboring Pasteurella in the respiratory passage during fatal infections of anaplasmosis, it was desired to observe the action of these agents in cattle suffering from a state of chronic infection of *A. marginale*. During May, 1935, calves 125 and 151 were exposed to Pasteurella through the nasal route as previously described. At this time, these animals were the recipients of 15 and 30 cc, respectively, of citrated blood representing three strains of bovine anaplasmosis. These animals reacted after the 18th day to a mild form of anaplasmosis and recovered their pristine condition in about ten days. Splenectomies were then performed, whereupon a state of chronic anaplasma infection ensued. During the chronic attack, over 50 per cent of the erythrocytes were parasitized and the hemoglobin content of the blood remained from 4 to 8 gm per 100 cc (Dare) during the period. Frequent blood cultures, stained blood smears and rabbit inoculations of blood from the affected animals during the period of chronic infection failed to show the presence of Pasteurella organisms. Frequent examinations of the bacterial flora of nasal secretions during the experiment showed the presence of *P. boviseptica*. Bull 125 died from the induced attack of chronic anaplasmosis after a period of four months. Cultures taken from the heart-blood a few hours after death showed the presence of *P. boviseptica*. Bull 151 was killed in an advanced stage of chronic anaplasmosis of two months duration. Cultures of the heart-blood, kidneys, liver and spleen and rabbit inoculations of heart-blood failed to reveal the presence of Pasteurella.

Results of the above experiments led to the conclusion that *P. boviseptica*, inhabiting the respiratory passage of cattle, appears in the blood shortly after death as a postmortem invader incident to *A. marginale* infection. *P. boviseptica* occurring in carcass material under these conditions may lead to an erroneous

diagnosis since such material is usually unsuitable for the demonstration of *A. marginale.*

Experiment 4: During September, 1935, opportunity was afforded for making clinical and postmortem observations in a herd of 60 dairy cows exhibiting clinical symptoms of catarrhal pneumonia and interstitial pulmonary emphysema following transportation by railway cars. The animals were purchased in the vicinity of Columbia, South Carolina. A number were held in shipping-pens for several days at the point of origin until the consignment could be completed and dispatched to Miami, Florida. Upon arrival at their destination, twelve animals were visibly affected, exhibiting emaciation, nasal discharge, cough, increased temperature and pulse, and dyspnea. A few were showing evidence of subcutaneous emphysema. During the four days following arrival, 13 additional animals included in this shipment developed similar though milder clinical symptoms. Eight animals in various stages of this condition were selected for the purpose of taking blood-cultures. Various quantities of blood were drawn under aseptic conditions into flasks containing Krack and Teasley's blood culture media and incubated at 37.5° C. Other blood-samples were collected in sterile test-tubes and held at room temperature for observations.

By these methods Pasteurella and colon-type organisms were secured from the blood-stream of one animal six hours before death. Blood-cultures from a second animal yielded *P. boviseptica* and a non-carbohydrate fermenting, Gram-negative rod. Cultures from a third animal secured eight hours before death yielded *P. boviseptica* alone. Postmortem observations made on these three animals shortly after death revealed a catarrhal bronchopneumonia with simultaneous interstitial pulmonary emphysema. Samples of heart-blood from these animals secured at autopsy revealed the presence of Pasteurella and colon-type organisms. The Pasteurella were pathogenic for rabbits in quantities of 0.001 cc of an 18-hour bouillon culture when given intraperitoneally. Blood-cultures from five animals taken during the early stages of the condition were sterile. No effort was made by the owner to isolate the recently shipped-in clinically affected animals from the native herd. The sick and well animals included in this shipment drank from a common trough and intermixed freely with the native cows without further spread of the disease. This would indicate that lowering of vitality incident to railway transportation is a serious predisponent, bringing about invasion of the blood-stream by Pasteurella and other organisms.

Experiment 5: Postmortem lesions in cattle, consisting of hemorrhages of the subcutaneous tissues, and of the great parenchymatous organs, serous membranes and gastro-intestinal edema, were observed by workers at the Florida Experiment Station[8-10] in *Crotalaria spectabilis* poisoning. Since these lesions may easily be suspected of having a septicemic origin, it seemed desirable to ascertain if *P. boviseptica* could be demonstrated in the blood of animals which had died of crotalaria poisoning. Three pounds of *C. spectabilis* seed were macerated in a menstruum of ammoniated water. The menstruum was decanted in 200-cc amounts, and given as a drench to a 300-pound bull at intervals of two or three days. The animal had previously been exposed to *P. boviseptica* by nasal spray. Symptoms of crotalaria poisoning developed after two weeks and no further menstruum was given. The bull died on the 29th day following the initial feeding. Four hours before death, blood-cultures secured under aseptic conditions did not show the presence of organisms. Postmortem examination a few hours after death showed petechial hemorrhages of the viscera and serous membranes, severe gastro-enteritis and edema. Samples of blood taken at autopsy revealed the presence of Pasteurella. In this case organisms were more numerous within the leukocytes in those blood-vessels situated nearest the laryngopharyngeal region. These organisms were highly virulent for rabbits, as shown by intraperitoneal inoculations.

Experiment 6: Pasteurella have been demonstrated as the etiological agent in some cases of omphalophlebitis of suckling calves.[1] Tests were run to determine if the organisms encountered in these investigations were capable of initiating symptoms and lesions of this condition. Six calves under 48 hours of age were secured from a herd where omphalophlebitis had not been known to occur. These calves were given umbilical injections using 2 to 3 cc of saline suspensions of various strains and densities of *P. boviseptica*. Following the injections, there occurred depression, inappetence, elevation of temperature, blood-flecked, foamy, fetid diarrhea, straining and emaciation, with occasional involvement of one or more joints. These symptoms were manifest over a period of several weeks, resulting in underdevelopment of the calves. Pronounced symptoms of bronchopneumonia developed in four of these calves about the 60th day following the umbilical injections.

Experiment 7: To test the virulence of *P. boviseptica* for the crow (*Corvus ossifragus*), 22 live specimens were trapped in the field and transferred to the isolation pens where they were con-

fined in wire cages. After being held ten days for observation, 0.1 cc of a 24-hour bouillon culture of Pasteurella, isolated from the bronchial secretion of an apparently healthy bovine, was instilled into the right eyes and nasal clefts of six crows. Fourteen hours later, three of the inoculated crows were dead and the other three were visibly sick. Forty-eight hours later, one uninoculated crow died. Several crows lived in the pen for a period of 66 days, exhibiting symptoms of chronic Pasteurella infection. Pasteurella were isolated from the blood-stream of those birds exhibiting acute and subacute types of infections. They were isolated from the posterior nares of the chronically affected birds at intervals during the experiment and from their blood-stream a few hours before death.

Postmortem lesions observed in these crows consisted of hemorrhagic laryngotracheitis, hemorrhagic enteritis, congested lungs, parenchymatous degeneration of the liver, spleen and kidneys, petechiae of the pericardium and increased pericardial fluid.

Summary

1. *Pasteurella boviseptica* is frequently encountered at autopsy in the blood and internal organs of Florida cattle.

2. Exposure of calves, yearlings and cows to *P. boviseptica* by means of ingestion and by nasal spray failed to produce septicemia.

3. *P. boviseptica* was recovered from the nasopharynx of cattle, showing no gross lesions, three months following mild exposure by means of nasal sprays and from the frontal sinus two months following introduction by means of trephining.

4. *P. boviseptica* may be demonstrated in the blood and internal organs of cattle shortly after death incident to *Anaplasma marginale* infection.

5. *P. boviseptica* may be demonstrated in the blood and internal organs of cattle autopsied following *Crotalaria spectabilis* poisoning.

6. *P. boviseptica*, colon-type and other organisms may frequently be isolated from the blood-stream of cattle showing advanced symptoms of bronchopneumonia and pulmonary emphysema which has developed incident to railway transportation.

7. Umbilical injections of *P. boviseptica* in suckling calves produce symptoms and lesions of omphalophlebitis.

8. *P. boviseptica* produced acute and chronic Pasteurella infection in the crow (*Corvus ossifragus*) when the organisms were

instilled into the nasal cleft or when the drinking water was contaminated.

9. *P. boviseptica* organisms encountered in these investigations were lethal for rabbits in amounts of 0.001 cc of a 24-hour salts bouillon culture given intraperitoneally or when a drop of the whole culture was instilled into the nasal passage.

10. The demonstration of Pasteurella in bovine carcass material of suspicious hemorrhagic septicema cases and its virulence for rabbits cannot always be accepted as a criterion regarding the etiological significance of such organism.

11. Realizing different degrees of virulence attributed to strains of Pasteurella under variable conditions, no evaluation is claimed for organisms other than those encountered and examined in these studies or for their action on animals under conditions other than those undertaken in the investigation.

ACKNOWLEDGMENTS

The author is indebted to Mr. Carlyle Carr, of the United States Biological Survey, for furnishing live specimens of the fish crow (*Corvus ossifragus*) used in these studies.

REFERENCES

[1]Leasure, E. E., Leinhardt, H. F., and Taberner, F. R.: Feline infectious enteritis. No. Amer. Vet., xv (1934), 7, pp. 30-44.
[2]Hutyra, F., and Marek, J.: Special Pathology and Therapeutics of the Diseases of Domestic Animals. (6th ed., Alexander Eger, Chicago, 1926), I.
[3]Moore, V. A.: Pathogenic and toxicogenic bacteria in the upper air passage of domestic animals. U. S. Dept. Agr., B. A. I. Bul. 3 (1893), pp. 38-48.
[4]Jorgenson, G. E.: Some studies of *P. bovisepticus*. Corn. Vet., xv (1925), pp. 295-302.
[5]Scott, J. P., and Farley, H.: Preliminary bacteriological report on shipping fever. Jour. A. V. M. A., lxxx (1932), n. s. 33 (2), pp. 173-186.
[6]Kracke and Teasley: Manual of methods for pure culture study of bacteria. Leaf. 2 (1933), p. 8.
[7]de Kock, G., and Quinlan, J.: Splenectomy in domesticated animals and its sequelae, with special reference to anaplasmosis in sheep. 11th and 12th Rpts., Dir. Vet. Ed. & Res., Union of So. Afr. (1926), pp. 369-480.
[8]Sanders, D. A., Shealy, A. L., and Emmel, M. W.: The pathology of *Crotalaria spectabilis* Roth poisoning in cattle. Jour. A. V. M. A., lxxxix (1936), n. s. 42 (2), pp. 150-156.
[9]Thomas, E. F., Neal, W. M., and Ahmann, C. F.: The toxicity of *Crotalaria spectabilis* Roth to livestock and poultry. Jour. Amer. Soc. Agron., xxvii (1935), 6.
[10]Becker, R. B., Neal, W. M., Dix, P. T., and Shealy, A. L.: A study of the palatability and possible toxicity of 11 species of Crotalaria. Jour. Agr. Res., 1 (1935), pp. 911-922.

DISCUSSION

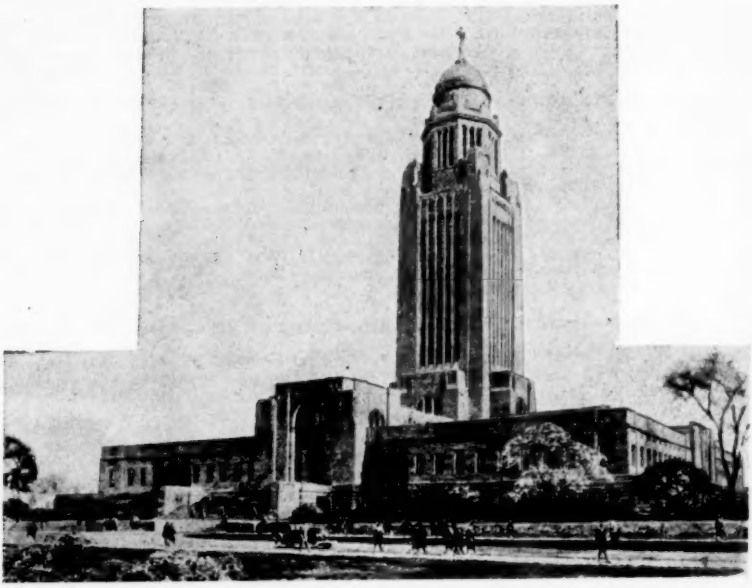
DR. BEAUDETTE: The question in my mind is whether Dr. Sanders took adequate measures to preserve the type of organism in the animal. In coöperative experiments with the Rockefeller Institute, on the epidemiology of "fowl cholera," we found that fulminating epizoötics frequently yield "fluorescent" organisms. These organisms injected in the nasal cavities of chickens regularly provoke a high mortality. This "fluorescent" type of organism is so designated because it does produce a large fluorescent colony on hemolyzed blood agar. However, if it is propagated on ordinary agar, or in broth not containing blood,

it very rapidly changes to what is known as the "blue" type—blue because when grown on media it produces a small, rather dry, blue, or slate-colored colony. This change is not reversible, that is to say, after numerous animal passages, or by propagation again on blood media, we have never been able to cause it to change to the fluorescent type. This blue type is also relatively avirulent, so that when inoculated in large doses into the nasal cavities, it rarely ever provokes death, and at most may produce only one of the localized conditions in the nasal cavity, or sinus.

It seems to me that in working with this infection in cattle, one should isolate the organism on media containing blood to preserve the type and then make the intranasal inoculations with an organism which has been propagated only on blood media, because if the organism is propagated on media that does not contain blood, it will readily revert—at least the chicken organism will—to the blue type which, when inoculated into animals, gives us at most only an occasional death or localized lesion.

DR. SANDERS: Personally I do not believe you can compare this organism in cattle with that in the chicken. My problem was to find out the significance of these organisms in the blood of cattle and I think we demonstrated that in the paper.

DR. C. P. FITCH: I think Dr. Sanders should be very highly complimented on this paper because it is of great significance. I would like to offer one suggestion, not only to him, but to anyone else who is working in this field, and that is a carefully conducted piece of work to determine whether or not there is a virus connected with these cases of shipping fever, from which the bovine hemorrhagic septicemia organism can also be isolated.



THE NATIONALLY FAMOUS NEBRASKA STATE CAPITOL AT LINCOLN, ONLY 60 MILES FROM OMAHA

THE IMPORTANCE OF ENDOTOXIN OF SALMONELLA AERTRYCKE IN THE DEVELOPMENT OF FOWL PARALYSIS*

By M. W. EMMEL, *Gainesville, Fla.*
Florida Agricultural Experiment Station

A paper was presented last year, before these sections, on the importance of hemocytoblastosis in the development of fowl paralysis. Although hemocytoblastosis is an essential process in the development of fowl paralysis, hemocytoblastosis in itself will not result in fowl paralysis unless certain other factors are associated with it. It perhaps should be stated here that hemocytoblastosis in the chicken is caused (1) by *Salmonella aertrycke* as well as certain other species of the genus *Salmonella.* (2) by the transmission agent which, in this study, is capable of inducing any of the leukemias when injected intravenously or intraperitoneally into susceptible birds, and (3) by certain other agents, some of which have an important bearing in this study and others of which are associated with entirely different disease processes.

It is the intention in this paper to discuss some of the properties of endotoxin derived from *S. aertrycke* as related to the development of fowl paralysis. The term endotoxin will be used to refer to the intracellular toxic substance of *S. aertrycke* prepared by tryptic digestion under favorable conditions and 1 cc of which is capable of killing a 20-gram mouse in from three to 16 hours. The latter represents the standard by which toxicity of endotoxin is usually measured.

Ever since Gaertner isolated the first known species of the paratyphoid group, *Salmonella enteritidis,* in Germany, in 1888, and demonstrated that a toxic substance could be derived from this microörganism, many investigations have been conducted on this and other microörganisms of this group to study the nature of this toxic substance. While the literature contains many conflicting results, it is commonly agreed that *S. aertrycke, S. enteritidis, S. shottmülleri, S. pullorum* and *S. suipestifer* each contain an endotoxin which is liberated only upon destruction of the cell.

Endotoxin has been found by most investigators to be thermostable, nondialyzable, antigenic, toxic and possibly protein in

*The data contained herein represent a portion of a thesis entitled, "The Mechanism of *Salmonella aertrycke* in the Development of Fowl Paralysis, Leukemia and Allied Conditions in Chickens," to be submitted to the faculty of Michigan State College in partial fulfillment of the requirement for the degree of Doctor of Philosophy. Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

nature. Many of the properties herein discussed, however, as related to fowl paralysis are being reported for the first time.

Of primary importance in this study is the neurotoxic action of endotoxin when injected intravenously into young chickens. Lameness, paralysis of both legs, and of the auditory nerve, wry neck, spastic cervical muscles and complete paralysis have been observed in some chicks following the intravenous injection of 1.0 cc of endotoxin into day-old chicks. Other chicks appear sleepy, droopy or apparently unaffected. The paralytic symptoms observed are transient, disappearing in from one to two hours, after which the bird becomes and remains apparently normal.

There is an age resistance in chickens to the neurotoxic action of endotoxin. While 1.0 cc of endotoxin is all that is necessary to induce temporary paralytic symptoms in many day-old chicks, the injection of increasing amounts is necessary to induce similar symptoms as birds progress in age. Approximately ten to 15 times the amount of endotoxin is necessary to induce such symptoms in birds six to eight months old, as is necessary in the case of baby chicks.

The repeated intravenous injection of endotoxin in amounts not capable of inducing paralytic symptoms results in mild hemocytoblastosis with many degenerative cell types as described in a previous paper on hemocytoblastosis. Endotoxin likewise *in vitro* is capable of destroying blood cells, leukocytes more readily than erythrocytes. The repeated intravenous, intraperitoneal and intracerebral injection of endotoxin over periods of time, as long as two months, fail to induce typical fowl paralysis as observed under naturally occurring conditions.

Previous reference has been made to endotoxin as being capable of inhibiting agglutination in the higher titres after incubation for twelve hours, when 0.1 cc of endotoxin is added to the standard tube test, immune serum and antigen of *S. aertrycke* being used. To reduce the titre of *S. aertrycke*-immune serum during incubation for 24 hours it was necessary to add from 0.025 to 0.075 cc of endotoxin. The inhibition of agglutination and reduction of titre is relatively proportioned to the potency of the added endotoxin. It is interesting that the endotoxin of *S. aertrycke* also inhibits agglutination and reduces the titre of other immune sera when homologous antigens are used as well as in the case of heterologous antigens in instances in which cross-agglutination occurs.

Endotoxin has also been found to inhibit phagocytosis. The ability of endotoxin to accomplish this is also relative to the potency of the product used. This phenomenon was also observed

by Wittenberg, Lederer and Mollov[8] in the case of the endotoxin of gonococci, and by Dudgeon, Panton and Wilson[1] in the instance of some of the other members of the paratyphoid group. The latter authors, noting considerable specificity in the reaction, suggested this means of differentiating certain bacteria.

The idea was advanced that if endotoxin held an important function in the development of fowl paralysis, its action should be evidenced in experiments in which a comparison was made between repeated injections of *S. aertrycke* alone *versus S. aertrycke* plus endotoxin. In the latter instance, 1.0 cc of endotoxin was added, as greater amounts often caused sudden deaths of birds from undetermined causes. In twelve experiments in which heterologous and homologous endotoxin of six strains of *S. aertrycke* were used, it was found that the causal microörganisms alone induced fowl paralysis in 22 of 108 birds six to twelve weeks of age, while the causal microörganism plus endotoxin induced fowl paralysis in 53 of 105 birds of the same age. No effort was made to induce as large a number of cases of fowl paralysis as possible; the experiments were merely placed upon a comparative basis. In the instance of added endotoxin the incubation period was shorter.

The second idea was advanced that if endotoxin holds an important function in the development of fowl paralysis, it should be possible to inject this product into day-old chicks, note those birds susceptible to its neurotoxic action, raise them to eight to twelve weeks of age, infect them then with *S. aertrycke* by repeated intravenous injections and note the differences in susceptibility to the causal microörganism at this age as compared to the difference in susceptibility to the neurotoxic action of endotoxin in the same birds as baby chicks. In experiments involving a total of 330 chicks, 158 showed paralytic or nervous symptoms when given an intravenous injection of endotoxin as baby chicks. After these same birds were raised to eight to twelve weeks of age, 148 developed fowl paralysis when exposed to repeated intravenous injections of *S. aertrycke*. Of extreme importance, however, is the fact that 137 of these 148 birds showed susceptibility to endotoxin as day-old chicks.

In other words, 86.7 per cent of 158 chicks susceptible to the neurotoxic action of endotoxin developed fowl paralysis as a result of exposure to the causal microörganism, when eight to twelve weeks of age, and only 0.64 per cent of the remaining 172 non-endotoxin susceptible chicks developed paralysis as a result of exposure to *S. aertrycke* when eight to twelve weeks of age. Many birds develop identical paralytic symptoms as the result

of endotoxin injection when baby chicks as when exposed to *S. aertrycke* when eight to twelve weeks of age.

SUMMARY

The intravenous injection of endotoxin of *Salmonella aertrycke* often results in transient but typical symptoms of fowl paralysis.

Birds show a progressive age resistance to the neurotoxic action of endotoxin.

Repeated intravenous injection of endotoxin in chickens results in mild hemocytoblastosis but repeated intravenous, intracerebral or intraperitoneal injections will not result in fowl paralysis.

Endotoxin in varying amounts *in vitro* inhibits agglutination and reduces the titre of *S. aertrycke*-immune serum and inhibits phagocytosis.

S. aertrycke alone induced fowl paralysis in 22 of 108 birds six to twelve weeks of age, while the same number of causal microörganisms plus 1 cc of endotoxin induced fowl paralysis in 53 of 105 birds of the same age.

By the injection of endotoxin into baby chicks it was possible to predict, with 86.7 per cent accuracy, the development of fowl paralysis when the same birds became eight to twelve weeks of age and exposed to repeated intravenous injections of *S. aertrycke*.

REFERENCES

[1]Dudgeon, L. S., Panton, P. N., and Wilson, H. A. F.: The influence of bacterial endotoxins on phagocytosis. Proc. Roy. Soc. Med., lxxxii (1910), p. 406.

[2]Dudgeon, L. S., Panton, P. N., and Wilson, H. A. F.: The influence of bacterial endotoxins on phagocytosis including a new method for the differentiation of bacteria. Proc. Roy. Soc. Med., lxxxiii (1910), p. 561.

[3]Emmel, M. W.: The etiology of fowl paralysis, leukemia and allied conditions in animals. I. Introduction. History and a bacterial theory of the etiology of these diseases. II. The intravenous injection of suspensions of *Salmonella aertrycke* in the chicken. Fla. Agr. Exp. Sta. Bul. 284 (1935).

[4]Emmel, M. W.: The etiology of fowl paralysis, leukemia and allied conditions in animals. III. The intestinal flora of chickens affected with enteritis associated with intestinal parasitism. IV. The pathologic manifestations of the causal microörganisms in the fowl. Fla. Agr. Exp. Sta. Bul. 293 (1936).

[5]Emmel, M. W.: The etiology of fowl paralysis, leukemia and allied conditions in animals. V. The oral exposure of chickens infected with various species of coccidia to *Salmonella aertrycke*. VI. The oral exposure of chickens infested with *Ascaridia*, *Taenia* and *Capillaria* to *Salmonella aertrycke*. Fla. Agr.[4] Exp. Sta. Bul. In press.

[6]Emmel, M. W.: The etiology of fowl paralysis, leukemia and allied conditions in animals. VII. Hemocytoblastosis in chickens as induced by *Salmonella aertrycke*. VIII. Hemocytoblastosis in naturally occurring cases of fowl paralysis, lymphomatosis and in other birds from the same flocks. Fla. Agr. Exp. Sta. Bul. In press.

[7]Emmel, M. W.: Hemocytoblastosis and its relation to the development of fowl paralysis and fowl leukemia. Jour. A. V. M. A., lxxxviii (1936), n. s. 41 (1), pp. 45-50.

[8]Wittenberg, J., Lederer, M., and Mollov, M.: Studies in phagocytosis. I. Effects of endotoxin on phagocytosis of gonococci. Jour. Immunol., xxv (1933), p. 135.

DISCUSSION

DR. E. P. JOHNSON: I would like to ask Dr. Emmel what criterion he uses for diagnosing these cases as fowl paralysis? Some of his pictures of the chicks indicate that those symptoms might be due to most anything. I have seen chicks with pullorum disease that looked just like some of those he has shown.

DR. EMMEL: The injection of endotoxin into baby chicks induces these transient symptoms, which are similar to those of fowl paralysis; however, there apparently are no microscopic or macroscopic lesions.

DR. JOHNSON: The symptoms you have produced, if there was no examination to show comparable lesions with what we get in the complex condition we speak of as fowl paralysis, would not necessarily indicate anything specific. Any disease, for instance, botulism, neighbors on very similar symptoms, but we do not confuse it with the condition we speak of as fowl paralysis.

DR. EMMEL: I do not claim that the injection of endotoxin induces fowl paralysis, but that symptoms similar to those observed in this disease are induced. I feel that endotoxin is an important feature in the mechanism of the causal microörganism in the production of typical fowl paralysis.

DR. JOHNSON: The legend on your slides conveys the idea that you were producing fowl paraiysis?

DR. EMMEL: I did not intend to convey that idea. The idea I wish to stress is that endotoxin, which is contained within what I feel is the causal microörganism for fowl paralysis, is capable of inducing paralytic symptoms similar to those observed in typical fowl paralysis.

DR. JOHNSON: Are you sure they are paralytic symptoms?

DR. EMMEL: What else could they be?

DR. JOHNSON: You might inject abortion organisms, for instance, and produce somewhat similar symptoms. Did you try any broth? The symptoms of stupor which you have shown might be simply due to foreign protein effect.

DR. EMMEL: Controls were injected with a mixture similar to the endotoxin preparation but minus the microörganisms essential in the preparation of endotoxin. I will agree with you that the injection of endotoxin into baby chicks is possibly not the only substance which one might inject into a baby chick and induce paralytic symptoms. There may be other substances. The other substances do not have any direct relationship to *Salmonella aertrycke*, the causal microörganism for fowl paralysis. However, the endotoxin injected has a close relationship to the causal microörganism; the endotoxin was extracted by digestion from this causal microörganism.

DR. JOHNSON: Did you try any other organisms besides *S. aertrycke*?

DR. EMMEL: The endotoxin of microörganisms of the *Salmonella* group, *S. aertrycke*, *S. enteritidis*, *S. schottmülleri*, *S. typhimurium*, *S. psittacosis*, and *S. pullorum* induce similar paralytic symptoms when injected into baby chicks. While we have not done a large amount of work on *S. pullorum*, we feel that some of the paralytic symptoms observed in outbreaks of pullorum disease in baby chicks and which are typical of those we have illustrated are caused by the endotoxin of *S. pullorum*. We feel that the reason fowl paralysis does not commonly occur following pullorum disease in chicks is because of the comparatively short period in which *S. pullorum* is circulating in the circulatory system. Either septicemia develops or the microörganism localizes in the tissues of the blood.

DR. JOHNSON: Do you consider that *S. pullorum* is a cause of fowl paralysis?

DR. EMMEL: Yes, but possibly not commonly in the field. The disease can be induced regularly experimentally with *S. pullorum*. Being a common pathogene for chickens this microörganism must be placed in a different category than some of the other members of the paratyphoid group.

DR. JOHNSON: How about *Eberthella typhi?*

DR. EMMEL: *E. typhi* induces the aleukemic types of this group of diseases.

DR. JOHNSON: Do you find that very consistently?

DR. EMMEL: We find it very consistently.

DR. JOHNSON: In that case this would be a public health problem?

DR. EMMEL: Yes, it is.

DR. W. R. HINSHAW: You spoke of *E. typhi* as a factor here. Are these organisms of fowl origin or human origin which you are using?

DR. EMMEL: Some of our critics have doubted our ability to identify microörganisms so we have taken steps to correct possible errors. *S. aertrycke* has been used as a basis for this study since this microorganism is more commonly encountered in naturally occurring outbreaks of this disease. *S. aertrycke* isolated by ourselves from chickens, and by other investigators from turkeys, pigeons and the human has yielded similar results. *S. aertrycke* secured from four different sources is capable of inducing fowl paralysis in chickens. Most of the strains, however, show a varying ability to induce the disease; this is probably due to a variance in pathogenicity. Strains of *E. typhi* of fowl and human origin both induce alenkemic types of this group of diseases.

DR. H. VAN ROEKEL: I would like to say something in connection with this symptom-complex which was illustrated by Dr. Emmel. I wish to report our observations concerning what we call "epidemic tremor" in chicks. We take some brain material from chicks which show symptoms of "epidemic tremor," and by emulsifying it in a saline solution and injecting the same in small amounts (0.1 cc), either intracerebrally or intravenously, we can produce symptoms similar to what Dr. Emmel has demonstrated. These chicks have not been examined histologically so I cannot report on that, but on gross observations no lesions were found.

This material obtained from the brain appears to be very toxic. It apparently did not occur in all brains examined. I think the toxic action of the saline-brain suspensions might help to explain the symptom-complex reported by Dr. Emmel.

DR. F. R. BEAUDETTE: Concerning Dr. Van Roekel's remarks regarding the toxicity of the brain, it might be well to point out that, a few years ago, Remlinger showed that brain tissue of certain animals was toxic for members of the same species. Anyone can demonstrate this quite easily by emulsifying the brain and inoculating animals not only intracerebrally, but intraperitoneally or intravenously, and in our experience from 25 to 50 per cent of the animals inoculated will die within three or four minutes. I believe it is known as "Remlinger's phenomenon." The nature of the phenomenon has not been determined.

Classroom Boner

Pupils being taught meanings of words not commonly used, came to dogma. Jimmy, told to form a sentence including the word, produced this—

"Our dogma had four puppies yesterday."

Animaldom.

STUDIES ON AN EPIZOOTIC IN DOMESTIC RABBITS CAUSED BY PASTEURELLA CUNICULICIDA*

By F. D. McKENNEY *and* J. E. SHILLINGER

Bureau of Biological Survey, U. S. Department of Agriculture
Washington, D. C.

INTRODUCTION

The prevalence of disease in wild and domestic animals caused by infection with Pasteurella organisms has been known for many years. Because of their wide dissemination and frequent isolation from apparently healthy tissues, there has at times been a tendency to diagnose vague and unstudied disease conditions as hemorrhagic septicemia. There is ample proof, however, that the Pasteurella organisms may at times be the cause of acute contagious diseases, as well as of more chronic conditions, and whenever encountered in the study of an epizoötic, their ability to assume a pathogenic rôle should not be overlooked.

It is the purpose of this paper[1] to record the presence of *Pasteurella cuniculicida* as a factor in a contagious disease of domestic rabbits; (2) to show that it may be pathogenic when inoculated into test animals by various routes; (3) to show the effect of the contagion in a colony of animals when no control measures were applied, and (4) to show the different manifestations of the disease, depending upon the relative susceptibility of infected rabbits.

Hemorrhagic septicemia infrequently assumes the form of a fatal epizoötic in commercial domestic rabbitries because the animals are usually maintained in single hutches, the opportunity for spread of the contagion thus being lessened. The disease has more often been considered as a cause of severe losses among domestic rabbits maintained for laboratory purposes. Under favorable circumstances, however, it has been found to spread through a commercial rabbitry, causing a maximum mortality of 40 to 70 per cent of the adult stock.

During the study of this disease, extending over a period of 2½ years, four distinct manifestations of the disease have been recognized: (1) the nasal type of infection, or purulent rhinitis, frequently associated with "snuffles"; (2) the acute, rapidly fatal septicemic form; (3) subcutaneous abscesses; and (4) the infection of the reproductive organs.

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LITERATURE

Smith,[1] one of the first to work with the disease in this country, described the causative organism and the septicemic form of the disease. De Kruif[2-5] later described the dissociation of the bacillus, but it remained for Webster[6-8] to determine the incidence of *Pasteurella cuniculicida* in epizoötics of rabbit respiratory infections among laboratory animals. As far as we are aware, there is no record of the disease existing as an infection of the reproductive organs, or of the fact that it could be transmitted by means of introducing the infection into the vagina. There is also a paucity of literature concerning the association of the infection with epizoötics of subcutaneous abscesses, as well as the incidence of mortality of this disease, based on experimental work, as it occurs in the commercial domestic rabbitry.

MATERIAL AND METHODS

During the period in which these studies were made, it was possible to follow the course of an epizoötic through its various stages and manifestations. Most of the material was obtained from one rabbitry, which usually maintains between 150 and 300 breeding does, while the number of young in the rabbitry varies between 50 and 1,500, depending upon the season of the year. Although *P. cuniculicida* had been isolated from animals in this rabbitry at previous times, the infection had not existed in epizoötic form until the spring of 1934, at which time certain management practices made it possible for the disease to spread rapidly.

Stock media used for the isolation of organisms during this work consisted of heart-brain infusion agar, serum-agar, and dextrose-brain broth. Identification of *P. cuniculicida* was accomplished by examination of a fresh broth culture for motility after which the non-motile organisms were run on carbohydrates and litmus, or brom-cresol milk.

SYMPTOMS AND GROSS PATHOLOGY

When the disease first assumed an epizoötic form, adult animals seldom lived longer than 36 hours after they were observed to be sick. The usual symptoms were refusal of food and water, lethargy, increased respiration, rough coat, and often a white purulent discharge from the eyes. The gross pathology in such cases was limited to congestion of the subcutaneous vessels, with an occasional case showing petechial hemorrhages in the subcutaneous fascia. The spleens were noticeably enlarged, soft and

pulpy. The lungs showed varying degrees of pneumonic changes, and usually a large portion of both lungs showed extreme congestion with early solidification. Cut sections revealed hemorrhagic, frothy mucus. There was almost invariably a hemorrhagic tracheitis and laryngitis as well as petechial hemorrhages over serous surfaces. Congestion and enlargement of lymph-nodes usually occurred.

As the epizoötic progressed, cases in which the disease was prolonged were common, and in this type the gross pathology was usually limited to solidification and caseation of both lungs in addition to a fibrinous pleuritis and pericarditis. Abscess formation in the submaxillary glands was also quite common, the abscesses varying in size from 1 to 10 cm in diameter. These subcutaneous abscesses, however, were not always confined to the submaxillary region, as in young animals they were found on the back, legs, umbilical region, and on the face. Those on the face were the result of the purulent exudate being impounded in the maxillary sinuses. The abscesses usually possess a heavy fibrinous wall, and it was quite common to find one large abscess with several smaller ones attached but separated by the thick abscess wall. In several instances these chronic infections assumed more of phlegmonous character, and cases were observed in which the entire subcutaneous tissue of the back from the last cervical to the first sacral vertebra was infiltrated with the characteristic exudate. A necropsy on animals affected with these subcutaneous abscesses rarely showed gross pathology except the regional enlargement. When the abscesses were opened, there was a typical creamy, very tenacious, thick exudate, without perceptible odor.

During the latter part of the epizoötic, the majority of cases observed were of the purulent rhinitis, or "snuffles," type. The most pronounced objective symptom in such cases was a sneezing, with an excessive nasal discharge which was usually mucoid during the first stages of infection but which rapidly changed to a purulent discharge. It was often possible to determine which animals were infected by the matting of the hair on the medial surfaces of the fore feet before other symptoms were evident. The tip of the nose often became congested and badly irritated so that a heavy persistent scab would form. These symptoms were in some cases accompanied by a purulent discharge from both eyes. Only a small percentage of such cases of infection were fatal unless complicated by other factors, but this type of the infection was of an intermittent nature. The nasal discharge would often disappear within ten days to two weeks, and the subject appear to be in good health for a week, after which time all

the symptoms would recur. A large number of rabbits showing the purulent rhinitis type of infection were autopsied, but, with the exception of a few cases, gross changes were found only in the upper air passages. The usual pathology was confined to an accumulation of purulent exudate in the maxillary sinuses, congestion of the turbinates, and varying degrees of laryngitis and tracheitis. Those cases that terminated fatally usually showed an extension of the respiratory infection to the lungs, resulting in pneumonia.

EXPERIMENTS

In determining the etiology of this epizoötic, several experiments were performed to test the transmissibility of the disease. Table I gives the results of inoculating animals with various types of material obtained from acute cases of septicemia.

In each case in which the animal died as a result of the inoculation, no difficulty was experienced in recovering the organism from all internal organs. The organism proved to be pathogenic when injected subcutaneously or intravenously, and regardless of the origin of the strain approximately the same response was elicited. During the early experiments, inoculated animals were chosen from a group in which the disease was not present, and a sufficient number of uninjected animals were in the unit to act as controls. In later experiments, however, it was not certain that the animals inoculated had not formerly been exposed to the infection and built up some resistance to the disease.

Rabbits that were inoculated by placing the infective material on the mucous membrane seldom died in less than six or seven days after being inoculated. The disease was not reproduced by dropping small numbers of the organisms into the nose, and when a larger dosage was employed there was danger of introducing the material into the lungs. The suspensions of the organisms used for this purpose were prepared by washing the growth from agar slants, and as the growth of this organism on such slants is not easily suspended in physiological saline, the suspension was about the same density as tube 3 of the McFarland nephelometer.

As the epizoötic progressed and the number of cases showing subcutaneous abscesses increased, a culture obtained from one of these abscesses was inoculated into a group of animals to determine its pathogenicity and to see if it was capable of producing the septicemic form of the disease. Table II gives the results of one of these experiments.

While the organism obtained from the subcutaneous abscess was pathogenic, the results would indicate that the organism was

Case	Source of Material	Date Injected	Method of Injection*	Died	Remarks
952	Discharge from eye of acute case of pasteurellosis	6–4–34	Swabbed into eye	6–12–34	Discharge from eyes on 6-11-34. *P. cuniculicida* recovered in pure culture
275	Suspension of culture from acute case of pasteurellosis	8–21	Subcut. 1.5 cc	1– 9–35	Multiple abscesses in peritoneal cavity
96			Intrav. 0.8 cc	8–29–34	Typical lesions of acute septicemia
276	Strain T. G-2 from acute case of septicemia in an 8-week-old animal	1–4–35	Intrav. 0.8 cc	1– 5–35	Pure growth of *P. cuniculicida*
277			Subcut. 1.5 cc	1– 5–35	Pure growth of *P. cuniculicida*
x880			Intran. 0.5 cc	—	No effect
278	Strain 256	1–4	Intrav. 0.8 cc	1– 5–35	Pure growth of *P. cuniculicida*
685			Subcut. 1.5 cc	8–14–35	Sacrificed. No gross lesions
x580			Intran. 0.5 cc	—	No effect
768	Purulent exudate from vagina in a case of acute metroperitonitis due to *P. cuniculicida*	6–25	Swabbed into vagina	—	Purulent vaginal discharge. *P. cuniculicida* isolated 7-3-35
659			Swabbed into vagina	—	Purulent vaginal discharge. *P. cuniculicida* isolated 7-3-35
x160			Swabbed into eye	—	No effect
x 50			Swabbed into eye	—	No effect
644			Intrav. 0.5 cc	7–25–35	Purulent fibrinous pleuritis and pericarditis. Lungs caseated
665			Intrav. 0.5 cc	8– 7–35	Sacrificed. No gross lesions
645	Lacrimal exudate from case of acute metroperitonitis	7–12	Swabbed into eye	—	7-22-35 no deleterious effect
632			Swabbed into vagina	7–19–35	Acute septicemia. *P. cuniculicida* recovered
645	Uterine exudate from transmitted case of *P. cuniculicida* infection	7–20	Swabbed into vagina	7–26–35	Acute septicemia. *P. cuniculicida* recovered

*Subcut. = subcutaneously; intrav. = intravenously; intran. = intranasally.

less virulent than those used in previous experiments. Although one animal died two days after an intravenous inoculation, the second animal lived for one month, and at necropsy was found to have multiple abscesses attached to the serous surfaces of the uterus, intestines, and the abdominal wall. The two animals receiving a subcutaneous inoculation did not die as a result of the inoculation. When killed, one showed a typical abscess at the point of inoculation from which the organism was recovered, while the second animal showed no gross lesions, and all cultures from this animal were negative for *P. cuniculicida*.

TABLE II—*Transmission experiments.*

CASE	SOURCE OF MATERIAL	DATE INJECTED	METHOD OF INJECTION	DIED	REMARKS
28			Intrav. 0.5 cc	10–23	Acute septicemia
40			Intrav. 0.5 cc	11–22	Multiple abscesses in peritoneal cavity
31	Strain from subcutaneous abscess (case 717)	10–21–35	Subcut. 0.8 cc	11– 3	Sacrificed. Subcutaneous abscess at point of infection. *P. cuniculicida* recovered
32			Subcut. 0.8 cc	11–24	Sacrificed. No gross lesions. Organism not recovered

In culturing the exudate from subcutaneous abscesses, it was found that cultures from some cases failed to yield a growth of *P. cuniculicida*. In order to determine the pathogenicity of the organisms present, and in an attempt to isolate the organism from a larger percentage of cases, guinea pig inoculation was used to supplement the usual culture methods.

Table III gives a résumé of a few cases of subcutaneous abscesses in which animal inoculation was used in addition to the usual culture technic. A quantity of the exudate was removed by means of a sterile transfer needle, or sterile swab, and suspended in physiological saline. From 2 to 5 cc of the suspension was then injected intraperitoneally into a guinea pig. At necropsy cultures were taken of the heart-blood and peritoneal exudate of the guinea pig in addition to direct smears of the peritoneal exudate.

Guinea pigs dead as a result of the injection showed enlargement of the spleen, without gross lesions, congestion and enlargement of lymph-nodes, and a diffuse, purulent, fibrinous periton-

itis. The liver and other organs were covered with a white exudate. In some cases there was acute congestion of the lungs with hemorrhagic tracheitis. The results of this experiment would seem to indicate that although the guinea pig is susceptible to the infection in some cases, there is only a slightly higher percentage of successful isolations by means of animal inoculation than by cultural methods alone, and that while it probably is of use in conjunction with cultural technic, it has few, if any, advantages over culturing numerous samples of the exudate.

TABLE III—*Results of attempts to isolate P. cuniculicida from cases of subcutaneous abscesses.*

CASE	LESIONS	CULTURES	GUINEA PIG INOCULATION	
			RESULTS	*P. cuniculicida*
688	No rhinitis. Abscess of submaxillary glands	Abscess positive	Pigs not affected	—
689	No rhinitis. Abscess of submaxillary glands	Abscess negative	Both pigs died 24 hrs.	+
690	No rhinitis. Abscess of submaxillary glands	Abscess negative	Pig 1 died 24 hrs. Pig 2 not affected	+
691	Abscess on right rear foot	Positive	Pig 1 died 24 hrs. Pig 2 died 2 days	+
692	Abscess below ear on side of face	Negative	Pigs not affected	—
695	Abscess in umbilical region	Abscess positive. Spleen negative	*	
709	Abscess posterior to left clavicle	—	Pig died 11 days	—
717	Submaxillary abscess	Positive	Pig died 24 hrs.	+
718	Submaxillary abscess	Positive	*	
719	Caseated uterine abscess	—	Pigs not affected	—

*Pigs not injected.

Success or failure in isolating *P. cuniculicida* from subcutaneous abscesses could be determined with a fair degree of accuracy by the character of the exudate. In those cases in which the abscess was developing rapidly in size and where the exudate was more fluid, *P. cuniculicida* could be isolated in practically every instance. In abscesses that had been present for some time and had probably undergone some resorption, however, the character of the exudate was usually of a thicker consistency, even tending to be caseous, and it was possible to isolate the causative organism in only a small percentage of cases.

Since it was found that guinea pigs were quite susceptible to the infection, it was decided to use these animals in isolating *P.*

TABLE IV—*Correlation of purulent rhinitis and bacteriological findings.*

CASE	LESIONS	CULTURES	INOCULATION	GUINEA PIG CULTURES	
				P. cuniculicida	OTHER ORGANISMS
25	Purulent exudate in sinuses. Turbinates congested	HB neg.	Pig 1 died 24 hrs. Pig 2 died 48 hrs.	+	—
655	Purulent exudate in sinuses	HB neg.	Both pigs died 48 hrs.	+	—
660	Purulent rhinitis	HB neg.	Both pigs died 24 hrs.	+	—
661	Purulent sinusitis	HB neg.	Pig 1 died 24 hrs. Pig 2 died 3 days	+	—
662	Nares filled with pus	HB neg.	Pig 1 died 48 hrs. Pig 2 died 3 days	+	—
663	Nares filled with pus	HB neg.	Both pigs died 2nd day	+	—
664	Purulent exudate in sinuses	HB neg.	Pig 1 died 24 hrs. Pig 2 died 3 days	+	—
669	Slight accumulation of purulent material in nares	HB neg.	Pig 1 died 16 days. No effect on 2nd pig	—	
670	Nasal discharge serous. No purulent material	HB neg.	Pig 1, no effect. Pig 2 died 2 days	+	+ *A. bronchisepticuis*
671	Excessive amount of purulent exudate in nares	HB neg.	Pig 1 died 24 hrs. Pig 2 died 48 hrs.	+	—
672	Sinuses filled with thick purulent exudate	HB neg.	Both pigs died 24 hrs.	+	—
673	Creamy purulent material in uterus. Sinuses filled with same material	HB neg. Uterus pos.	Pig 1 died 24 hrs. Pig 2 died 48 hrs.	+	
675	Sinuses filled with purulent exudate	HB neg.	Pig 1 died 2 days. Pig 2 died 3 days	+	—
676	Sinuses filled with purulent exudate	HB neg.	Both pigs died 2 days	+	—
677	Purulent exudate was found in uterus. Nares clear	HB pos. Exudate neg.	Pigs injected with uterine exudate. Both died 24 hrs.	+	—
678	Abscess in mediastinum. Sinuses filled with purulent exudate	*P. cuniculicida* from abscess. HB neg.	Both pigs died 24 hrs.	+	—
679	Previous rhinitis. Sinuses clear	None	Both pigs died 24 hrs.	+	+ *A. bronchisepticus*
680	Purulent exudate in sinuses	HB neg.	Both pigs died 2 days	+	—
681	Sinuses filled with purulent exudate	HB neg.	Both pigs died 24 hrs.	+	—
682	Muco-purulent exudate from nose	HB neg.	Both pigs died 48 hrs.	+	—

TABLE IV—*Correlation of purulent rhinitis and bacteriological findings—Concluded.*

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CASE	LESIONS	CULTURES	INOCULATION	GUINEA PIG CULTURES	
				P. cuniculicida	OTHER ORGANISMS
683	Sinuses filled with thick purulent material	HB neg.	Both pigs died 48 hrs.	+	—
684	Left sinus filled with purulent exudate	HB neg.	Both pigs died 36 hrs.	+	—
685	Slight purulent rhinitis. Air-passages clear	HB neg. Streak plate fr. nose, *A. bronchisepticus*	Pigs not affected	—	—
686	Both sinuses completely filled with thick purulent exudate	HB neg.	Pig 1 died 24 hrs. Pig 2 died 2 days	+	—
697	Sinuses clear. Congestion of turbinates. Rhinitis	*A. bronchisepticus* fr. nasal swab	None inoculated	—	—
698	Right sinus filled with purulent exudate	*P. cuniculicida* fr. sinus	None inoculated	—	—
699	Both sinuses filled with thick purulent exudate	*P. cuniculicida* and *A. bronchisepticus* fr. streak plate	—	—	—
706	Uterus abscessed	*P. cuniculicida* fr. abscess	—	—	—
708	Purulent rhinitis	None	Guinea pig died 8 days	—	—

cuniculicida from the nasal discharge of rabbits affected with the purulent rhinitis type of the disease. It was thought advisable to determine the percentage of such cases harboring *P. cuniculicida* that showed no other symptoms than a nasal discharge. Although animals showing such symptoms seldom died as a result of the infection, provided there were no complicating factors, it had been demonstrated that the organism obtained from some of these cases was pathogenic. Consequently, if *P. cuniculicida* was present in many of the animals showing a purulent rhinitis, it would have considerable influence on recommendations as to the control of the disease, as such cases would constitute a source from which the infection might spread.

It was also believed that by injecting the material obtained from the nasal passages of affected rabbits into guinea pigs and culturing the heart-blood of the pigs dying as a result of the inoculation, it would eliminate those organisms usually found in the nasal flora of domestic rabbits, but which lack invasive properties, thereby making the isolation of *P. cuniculicida* less laborious. At the same time the material was obtained for guinea pig inoculation, a culture was taken from the heart-blood of the affected rabbit to determine if the organism might be present in the blood-stream. Those animals that were not killed in the experiment were cultured by introducing a small sterile swab into the nasal passage and slowly revolving it in an effort to touch as much of the surface as possible. The swab was then streaked on agar plates, after which it was macerated with physiological saline solution for guinea pig injection. Animals to be killed were destroyed by injecting a few cubic centimeters of air into the marginal ear vein. The skin was then removed from over the face with instruments which were placed in boiling water while not in use. The nasal passages and sinuses were then exposed by cutting through the overlying bones and turning them back with heavy shears. A quantity of the purulent exudate from the sinus or nares was picked up by means of a sterile swab which was macerated with sterile saline and the resulting suspension injected intraperitoneally into two guinea pigs, the first pig receiving 4 cc and the second pig 1 cc of the suspension.

Of the 29 animals showing evidence of a purulent rhinitis, there were only eight in which *P. cuniculicida* was not recovered either by means of guinea pig inoculation or by direct streak plate. Another interesting feature of this experiment was the evident localization of the infection, as in no instance was the organism obtained from the heart-blood of the rabbit unless there were lesions in parts of the body other than the upper respira-

tory tract. The infection, when confined to the nasal passages, was present as a localized chronic disease which may become pathogenic for the host under favorable conditions. It is recognized that in some cases *Alcaligenes bronchisepticus* may give rise to similar conditions, but in this epizoötic *A. bronchisepticus* did not play a prominant part. The majority of guinea pigs, regardless of the difference in dosage, died within 48 hours. Special precautions were taken to autopsy the pigs dying as a result of the inoculation, as soon as they were reported dead.

Discussion

Young animals and mature bucks were only infrequently affected with the acute septicemic form of the disease, but the disease seemed to take the form of a purulent rhinitis or subcutaneous abscesses in these animals. In one experiment the infection was introduced into two colonies of young females. There were 25 animals in each colony, all about four months of age, with many litter mates being divided between the two colonies. These were all fed and cared for under the same conditions. In lot 1, no animals were removed from the pen, although showing symptoms of the disease; while in lot 2, any animal showing evidence of infection with *P. cuniculicida* was immediately isolated. In lot 1, nine of the 25 animals died as a result of *P. cuniculicida* infection; while in lot 2, only one animal died as a result of the disease. This indicates that if careful observation is made of animals that are exposed to the infection it is possible to control the disease to a large extent by sanitary precautions. It further indicates that this class of animals is relatively insusceptible to the infection, as in the lots where no effort was made to control the disease only 36 per cent of the animals died as a result of the infection.

During the early stages of the epizoötic, history of the animals affected strongly indicated that the disease spread by contact from hutch to hutch. While possibilities of the animals coming into contact with the infection through other means could not be entirely eliminated, the disease at this time was confined to a small area in the rabbitry, and animals outside this area that had not come into definite contact with infected animals were not, as yet, being affected. Later, when the infection had become widespread, it was impossible to prove by what means the disease was being disseminated, although in a group of young animals, all in separate hutches, it was regularly observed that cases of purulent rhinitis frequently spread to adjoining hutches. These animals were not being handled, and all feed came from a supply

used for feeding this group only. It was not unusual for an animal to show a purulent discharge from the nose with the hair on the fore legs matted, and then a week or ten days later the animal in the adjoining hutch to show similar symptoms.

It is possible to transmit the various forms of pasteurellosis in domestic rabbits by using infective material from nasal secretions, subcutaneous abscesses, and the discharge from the eyes, blood, or suspensions of the spleen or liver from acute generalized cases of infection. Cultures of the organisms from infective material will also frequently produce the disease, but there seems to be no specificity in origin of the material or culture, as the organisms obtained from a chronic, localized infection often produce the acute, septicemic form of the disease, depending probably upon the susceptibility of the inoculated animal. In substantiation of Van Es'[9] observations, it was found that by using a culture of proved virulence it is possible to reproduce the acute type of the disease, but it is by no means certain that such transmission can be repeated indefinitely. Consequently, in testing the transmissibility of infections due to *P. cuniculicida* it has been necessary to use larger series of animals from non-infected groups for test purposes in order to decrease the probability of encountering unusual resistance or immunity to the infection.

It would seem that the physical condition of an animal is a greater factor in the individual resistance to the disease than any resistance which might be acquired through coming into contact with the infection. The greatest mortality due to acute *P. cuniculicida* occurred in pregnant females, the majority of fatalities occurring within three or four days before or after birth of the young. Mature males were very infrequently affected with the acute septicemic form of the disease.

Data on the percentage of the animals affected during the epizoötic could not be obtained because of the great fluctuation in the number of animals present in the rabbitry. It was estimated that at least 70 per cent of the adult and junior animals were affected with some form of the disease.

The susceptibility of guinea pigs used for inoculation purposes seems to be quite constant, and although the susceptibility of chickens to the infection has not been tested as yet, in one rabbitry where the acute form of the disease was prevalent, chickens, turkeys and ducks were permitted to run in the rabbitry, eating the discharge from affected animals as well as the carcasses of animals that had died with the disease, without deleterious effect.

In an effort to control an epizoötic of pasteurellosis in a domestic rabbitry, cultures of various forms of the disease were submitted to a biologic laboratory for the making of a killed culture vaccine. The vaccine was administered subcutaneously in two injections, a first injection of 1.5 cc, and then from 7 to 10 days later, a second injection of 5 cc being administered. As those in charge of the rabbitry were interested only in controlling the disease, it was not possible to hold a sufficient number of uninjected animals as controls, but, while use of vaccine may have increased to a slight degree the resistance of the animals to the infection, it did not protect them against the disease. Since in the course of epizoötics the infection may decrease in virulence, it cannot be stated that benefit resulted from the use of the vaccine. Control measures, therefore, must be based on sound sanitary principles in addition to such measures as may help to increase the resistance of the animals. Inclusion in the diet of an adequate amount of vitamin A should not be overlooked. On account of the rapid deterioration of cod-liver oil, in addition to the fact that there is some question as to the tolerance of herbivora for this product, it is thought advisable to use succulent green food as a source of this vitamin whenever it is possible to obtain such material. Many rabbit breeders object to the use of succulent green food on the basis of their belief that it causes intestinal parasites. When it is explained to them, however, that such material has a food value in addition to being a source of vitamin A, their objections can usually be overcome. From the standpoint of health, it is a better practice to use a small amount of succulent green vegetation in the daily ration than to use larger amounts at irregular intervals. Destruction or isolation of affected animals, with adequate disinfection of contaminated equipment, seldom fails to keep the disease under control.

Summary and Conclusions

An epizoötic in domestic rabbits in which *Pasteurella cuniculicida* was isolated as the causative organism has been recorded. Three forms of the infection were recognized: (1) an acute, rapidly fatal, septicemic form; (2) a subacute nasal rhinitis form; and (3) a chronic type expressed by subcutaneous abscesses. Cultures from all three types were used in transmitting the disease, but the type produced depends on the individual resistance of the animal and not on the source of the infective material. There is no certainty that such transmissions can be repeated indefinitely.

References

[1]Smith, T.: Jour. Comp. Med. & Surg., viii (1887), p. 24.
[2]DeKruif, P. H.: Jour. Exp. Med., xxxiii (1921), p. 773.

[3]DeKruif, P. H.: Jour. Exp. Med., xxxvi (1922), p. 309.
[4]DeKruif, P. H.: Jour. Exp. Med., xxxv (1922), p. 561.
[5]DeKruif, P. H.: Jour. Exp. Med., xxxv (1922), p. 621.
[6]Webster, L. T.: Jour. Exp. Med., xxxix (1924), p. 837.
[7]Webster, L. T.: Jour. Exp. Med., xxxix (1924), p. 843.
[8]Webster, L. T.: Jour. Exp. Med., xxxix (1924), p. 857.
[9]Van Es, L.: The Principles of Animal Hygiene and Preventive Veterinary Medicine. (John Wiley & Sons, New York) p. 473.

Treatment of the Dog

By ROBERT STEVENSON

All you who own a faithful friend,
These words of mine do ponder;
Don't call a quack, his aid to lend,
Lest from your pet you sunder.

But if your dog does droop and wail,
And shows some bad distresses,
Then call a doctor without fail,
Depend not on caresses.

Despise the use of patent food,
Or any such noxious ration.
Feed beef and lamb, for they are good,
For strength and animation.

To him much exercise afford;
It stimulates digestion,
And to him great liberty accord,
It relieves him of depression.

Vile nostrums shun; do not heed
Those who loudly advertise,
As selfish aims and lure of greed
Foster every sordid enterprise.

So now, dear people, use common sense;
No dog is a vegetarian,
Do not regard this an offense;
Just call in a veterinarian.

(*Editor's note:* Mr. Stevenson has been kennelman at the University of Pennsylvania Veterinary Hospital since 1898.)

74th ANNUAL CONVENTION A. V. M. A.

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STREPTOCOCCIC INFECTIONS IN DOGS
I. "Acid Milk," Arthritis and Post-Vaccination Abscesses*

By H. J. STAFSETH, W. W. THOMPSON *and* LISA NEU
Department of Bacteriology, Michigan State College
East Lansing, Mich.

Broadhurst[1] states that streptococci inhabit the alimentary tract of cows, horses, cats and dogs as commonly as they do that of man. Holman[2] classified, on the basis of their action on blood, lactose, mannite, salicin and sucrose, a large number of strains of streptococci isolated from dogs and supplied by Broadhurst. According to this classification all but one strain, *Streptococcus equinus*, were identical with human streptococci. The following names were used for the streptococci isolated from dogs: *Streptococcus infrequens, Strep. pyogenes, Strep. anginosus, Strep. fecalis, Strep. mitis, Strep. salivarius, Strep. nonhemolyticus* II, *Strep. nonhemolyticus* III, *Strep. equinus, Strep. ignavus.* Thus not a single one of these organisms is taken to be a real canine streptococcus. In view of the work of Edwards,[3, 4] Lancefield,[5] Plummer[6] and Edwards,[7] Holman evidently employed inadequate differential methods, since these investigators have shown that human and animal streptococci can be differentiated by fermentation and serological methods. Zinsser and Bayne-Jones[8] refer to dogs as being rather refractory to experimental streptococcus infection. No reference is made to natural streptococcic infections, although such infections of cats, mice, guinea pigs and chickens are mentioned. Rice[9] states that young dogs and young rabbits seem to behave most like the human species but are far from ideal test animals. This is in connection with experimental work in animals with human streptococci.

Veterinary literature is conspicuously lacking in regard to streptococci and streptococcic infections in dogs. Our work with this problem is not extensive and certainly not complete, but we believe that what we have found is of sufficient interest and significance to warrant presentation at this time.

MATERIAL AND METHODS

The source of our material was of three kinds, namely: apparently healthy males and females, diseased adults, and dead or moribund pups. The pups were examined mainly for the pur-

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pose of ascertaining the cause of pup mortality, thought by many dog-raisers and even veterinarians to be due to acid milk. After we had obtained some evidence suggesting that so-called acid milk is an infectious disease, cultures were made from the vagina, mammae and milk of females and the prepuce of males to determine if the organisms obtained from the various organs of the pups might have been contracted from adult carriers, more especially the dams.

Our methods were the usual bacteriological procedures with the following innovations: Instead of the usual beef infusion we used chicken infusion. In making this infusion we simply remove the skin, intestines and abdominal fat from chickens brought in for autopsy, provided they are in good flesh, grind them, bones included, and using the usual proportions of meat and water, obtain our infusion by steaming in flowing steam for two hours. The can in which the infusion is made is a cylindrical one, on the bottom of which is a perforated false bottom to which is fastened a hollow handle. When the meat has been coagulated by heat, it can be removed simply by lifting the false bottom by the handle, which is long enough to extend above the contents of the can and at the same time short enough to permit a cover to be placed on the can. In making agar from this infusion we use 1 per cent neopeptone (Difco) and, when blood-agar is wanted, 5 per cent sheep blood. The reaction of the medium is pH 7.4. One per cent neopeptone chicken infusion broth, pH 7.4, containing ground brain in such proportions that there will be at least 2 cc of broth above the brain tissue, is used in growing microaerophilic organisms or organisms which require some other physicochemical condition found in such a medium.

We now use chicken infusion and neopeptone for all our plain media, broth and agar, when working with organisms that do not grow well on ordinary meat extract or infusion media.

The following broth, a slight modification of a medium described by Devereux,[10] is used for obtaining more luxuriant growth of streptococci than can be obtained in most common media:

Neopeptone	5 gm
Yeast extract (Difco)	5 gm
Peptonized milk (Difco)	5 gm
Salt	5 gm
Dextrose	10 gm
Water	1,000 gm

Adjust to pH 7.4 and autoclave for 15 minutes at 15 pounds.

In making isolations of streptococci and other organisms from the throat, nose or vagina, or any other source where we have to

do with mucous material, we make streaks on blood plates and at the same time inoculate a plain neopeptone broth tube and a neopeptone brain broth tube. In about 18 to 24 hours, streaks are made on blood plates from the two broth tubes and it has been found that, especially in work with humans, certain organisms can be more easily detected and isolated in this way than by a direct plating method. For example, a good separation of *Staphylococcus aureus* and streptococci may be obtained in this way when, on the direct plate, the staphylococcus may be missed. Generally the staphylococcus is obtained from the plain broth tube. Individual colonies are picked and transferred to blood-agar slants if the organisms seem to be of the kind that require blood. In order to accustom them to growth in the absence of blood, it has been found useful to pass them through one or more cultures of plain neopeptone broth before planting them on plain agar.

Our fermentation media contain 1 per cent peptone and 0.5 per cent salt, with 1 per cent Andrade's indicator and 1 per cent of the respective sugars. Heavy inoculations are made to insure growth and the tests are repeated in doubtful cases to check the results.

Occurrence of Streptococci in Dogs

Our work was done with the specific purpose in mind of determining the relationship of streptococci to certain pathological conditions, rather than to see just how extensively streptococci occur in the various organs and anatomical parts of dogs.

Table I—*Occurrence of streptococci in dogs.*

Age and Sex	Animals Examined	Animals Yielding Streptococci					Animals Yielding No Streptococci	Carriers (%)
		Alpha	Beta	Gamma	Atypical*	More Than One Type		
Adult females	19	0	13	1	4	2	3	84.2
Adult males	5	0	1	1	1	0	2	60.0
Adults (Sex unknown)	2	0	1	2	2	2	0	100.0
Pups	18	2	7	0	1	2	10†	44.4
Totals	44	2	22	4	8	6	15	65.9

*Initial green zone changing to clear hemolytic zone, or hemolytic zones with faintly greenish tint, or green zone surrounded by hemolytic zone, or hemolytic zone next to colony, then narrow green zone, then narrow hemolytic zone.

†Six from one litter and four from another.

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Adult males	5	0	1	1	1	0	2	60.0
Adults (Sex unknown)	2	0	1	2	2	2	0	100.0
Pups	18	2	7	0	1	2	10†	44.4
Totals	44	2	22	4	8	6	15	65.9

*Initial green zone changing to clear hemolytic zone, or hemolytic zones with faintly greenish tint, or green zone surrounded by hemolytic zone, or hemolytic zone next to colony, then narrow green zone, then narrow hemolytic zone.

†Six from one litter and four from another.

With the exception of the pups which were autopsied, our examinations were limited to such localities as the throat, nares, mammae, vagina, prepuce, abscesses and skin lesions. On two occasions feces and milk also were examined.

Table I shows that, in such examinations, one encounters streptococci rather frequently in dogs.

Table II gives the kind of dog and the anatomical regions or nature of specimen from which the different types of streptococci were isolated.

Tables I and II show that streptococci occur commonly in dogs. They show also that the hemolytic or beta type is encountered more frequently than the others in the anatomical regions and

TABLE II—*Sources of the different types of streptococci.*

Dog	Description	History	Source and Type of the Streptococci Isolated			
			Alpha	Beta	Gamma	Atypical
1	Chesapeake Bay female	Lost pups				Feces[1]
2	Chesapeake Bay female	Lost pups				Feces
3	Bull dog male	Hospital case			Ulcer on leg	
4	French bull	Lost pups		Vagina[2]		
5	French bull pup	Died 3rd day[3]	Heart	Liver[2]		
6	French bull pup	Died 3rd day		Heart, lungs, kidneys, intestines[2]		
7	Adult female	From dog pound		Vagina[2]		
8	Collie female	From dog pound. Metritis		Vagina[2]		
9	Shephard female	Dog pound		Vagina[4]		
10	Male	Dog pound		Prepuce after mating with No. 7; none before[4]		
11	Pup	Died (distemper)[5]		Vagina[4]		
12	Hound female	Diabetes		Vagina[4]		
13	Cocker spaniel	Lost litter, 6 pups, 3rd day		Vagina[4]		
14	Boston bull	Lost pups		Vagina	Vagina	
15	Chesapeake Bay female	Died few days old		Lung[4]		
16	Chesapeake Bay pup	Died few days old	Same litter	Peritoneal cavity, heart, lungs, intestines[6]		

TABLE II—*Sources of the different types of streptococci—Continued.*

DOG	DESCRIPTION	HISTORY	SOURCE AND TYPE OF THE STREPTOCOCCI ISOLATED			
			ALPHA	BETA	GAMMA	ATYPICAL
17	Chesapeake Bay pup	Died few days old		Right femorotibal articulation[7]		
18	Chesapeake Bay pup	Died few days old				Lung
19	No description given	Skin abscess		Pus[2]	Pus	Pus
20	Pup	Died few days old	Lung	Blood, pericardium, liver, umbilicus[8]		
21	Cocker spaniel	Had lost litters				Vagina
22	Cocker spaniel	Lost 1 pup from 2 litters		Vagina		
23	Cocker spaniel	Recently lost pups at 3 weeks		Vagina		
24	Cocker spaniel	Lost 1 litter		Vagina		Vagina
25	Cocker spaniel	In whelp now; lost last pups		Vagina		
26	Cocker spaniel	Lost some pups		Vagina[4]		
27	Cocker spaniel male					Prepuce
28	None given	Abscess after rabic vaccination			Pus	Pus
29	Doberman	Lost pups		Vagina[2]		

[1]Vaginal swabs not taken. Fecal sample sent in.
[2]Typical fermentation reaction. Broth clearing, fine granular sediment.
[3]Third day after birth.
[4]Typical fermentation reaction. Broth clearing, coarser sediment.
[5]Symptoms suggested distemper.
[6]Culture not identified.
[7]Typical fermentation reactions, appearance of broth cultures not recorded.
[8]Typical fermentation reaction, slightly different colony. The rest, those not marked by [2, 4, 6, 7 or 8] show fermentations differing from those of the marked ones.

lesions examined in this series. A little more than 68 per cent of the females and 39 per cent of young pups, several of them not three days old, harbored this type. The percentage of infection encountered in pups may be misleading because ten of the ones yielding no streptococci came from two litters, six from one and four from another. Fourteen of the 22 hemolytic cultures isolated seemed to represent one species or at least two very

closely related species or varieties. The common occurrence and invasiveness of this organism suggested that it might be well to concentrate our efforts on the determination of its identity and true significance, leaving the others for future work.

Morphology and Cultural Characteristics of the Type in Question

This organism is a Gram-positive streptococcus from 0.9 to 1 micron in size, occurring singly, in pairs, but more frequently in short or long chains. It varies somewhat in arrangement and shape in various media. There may be well-rounded forms and also elliptical elements or even rods in a single chain. In broth regular Gram-positive cocci in long chains predominate.

This organism is as a rule very hemolytic and prefers blood media for continued growth. It will grow on neopeptone chicken infusion agar, but it is not easy to keep it alive for any length of time unless blood media are used. The colonies vary from 0.5 to 1 mm in size. They are convex, smooth, glistening, faintly yellowish white, opaque, with entire margins. The hemolytic zone surrounding the colonies may vary from a narrow one to 5 mm in diameter. In neopeptone chicken infusion broth it grows well with clearing and sedimentation. The sediment varies from a finely granular to a somewhat more coarsely granular one.

Fermentation Reactions

Table III shows the characteristic fermentation reactions of four representative strains. These strains were sent to Dr. P. R. Edwards, of the University of Kentucky, who kindly checked their behavior in lactose, sucrose, mannite, sorbitol and trehalose. He also checked two of them serologically and is of the opinion that this organism belongs to group C of Lancefield, *i. e.*, the animal pathogens. In our fermentation studies we have included dextrose, maltose and salicin, because some of the other varieties of streptococci isolated from dogs differ in their actions on these sugars.

Habitat, Mode of Spread and Pathogenicity

It appears that the natural habitat of this organism is the female genital tract. We have not isolated it from the genitals of males except in one case in which a male was mated to a female carrier. This male carried it in his prepuce for at least four days after mating. An examination of his prepuce 15 days after mating revealed no streptococci. The female to which this dog was mated did not yield streptococci from the vagina while pregnant

TABLE III—*Fermentation reactions of four representative strains of the streptococcus under investigation.*

STRAIN	SOURCE OF STRAIN	DEXTROSE	LACTOSE	MALTOSE	MANNITE	SALICIN	SUCROSE	SORBITOL	TREHALOSE
I (8)	Prepuce of male[1]	+	+	+	—	+	+	—	—
II (5)	Vagina of collie[2]	+	+	+	—	+	+	—	—
III (2a)	Liver of pup[3]	+	+	+	—	+	+	—	—
IV (4)	Vagina of mongrel[4]	+	+	+	—	+	+	—	—

+ = acid formation; — = no fermentation.

[1]Infected by mating to a female carrier (4).

[2]This female was obtained from a dog catcher. She had recently whelped and was suffering from metritis (bloody, purulent discharge from vulva) at time of examination.

[3]Died on third day after birth. This pup's mother carried this organism in her vagina.

[4]In heat at time of examination. Mated to male[1] immediately after examination.

but did so again after having whelped. She was examined during her next estrum, was found to carry this streptococcus, whereupon she was again bred by the above-mentioned male, who this time also picked up the organism.

Since this organism has been isolated from the pus from abscesses in an adult dog and from the umbilicus, intestines, heart, lungs, kidneys, vagina, blood, peritoneal cavity and the femorotibial articulation of pups, it does seem that it has definite earmarks of being a pathogen.

GROSS PATHOLOGICAL CHANGES

The following pathological changes have been observed with marked regularity in pups: swollen and inflamed inguinal and prescapular lymph-glands. In one case the right femorotibial articulation was inflamed. There were abscesses in the foot pads and interdigital spaces of two pups. One pup also showed skin abscesses at the time of examination. This was true also of the abscesses in the foot pads. These had been drained. The pups showing these abscesses were three weeks old. Changes observed in these pups as well as in younger ones were: pericarditis, some cellular infiltration in the pericardium (older pups), enlargement of the spleen and kidneys, enteritis, pulmonary congestion and sometimes pneumonia. The kidneys also were very congested. In two pups, three weeks old at the time of the autopsy, there was impaction of the colon with rather dry fecal material.

Streptococci in Skin Lesions

The streptococcus just described as well as the viridans and gamma types, have been isolated from abscesses and ulcerations of the skin. One of these cases seems worthy of mention. It had to do with a valuable dog which developed abscesses and died following rabies vaccination. The abscess formation started at the point of vaccination and streptococci were isolated from the pus. No streptococci were obtained on repeated culturing of a sample of vaccine of the same kind as that used for this dog. The vaccine was not sterile, however, a nonhemolytic diphtheroid being isolated by first inoculating broth, incubating it for 18 to 24 hours and then transplanting the organism to the blood plates.

The streptococcus isolated from the pus of the vaccinated dog was a Gram-positive, small, regularly shaped streptococcus forming long chains in broth cultures. The colonies on blood plates were of the size of a small pin-head, irregular in shape, grayish, shiny and somewhat rugose. Next to the colony was a hemolytic zone, then a narrow green zone and then again a narrow hemolytic zone. When passed through broth prior to being plated on agar, the colonies were smooth, convex, grayish, shiny, opaque and were surrounded by a hemolytic zone. This organism fermented dextrose, lactose, maltose, attacked sucrose but slightly and did not ferment mannite and salicin. There was also another nonhemolytic, Gram-positive coccus, arranged in pairs, clusters and a few short chains. This organism gave rise to convex, grayish white, opaque colonies of the size of a pin-head. Unfortunately we did not have a chance to examine the carcass of the dog. We do not know which one of the organisms isolated might have been responsible for the death of the animal, nor indeed that any one of them was responsible. However, in view of the fact that streptococci do occur frequently in skin and muscular lesions of dogs, it may be well to employ caution in surgical procedures and especially in rabies vaccination since the nature of the vaccine and the amount injected are such as to cause mechanical injury and the formation of cold abscesses at least. Since dogs harbor streptococci in various locations of their bodies and perhaps also on the skin, it is not difficult to imagine what could develop in the line of secondary infection.

Articular and Muscular Rheumatism*

In the winter of 1932, we were asked to make a bacteriological examination of a six-year-old doberman pincher (male) which was suffering from an ailment that had been diagnosed as articu-

*These cases are not included in Tables I and II.

lar and muscular rheumatism. We believe that polyarthritis might have been a better name for this particular case. In the winter of 1931, this dog became ill and his malady was diagnosed as indicated above. The joints in his legs were swollen and painful. He showed some stiffness throughout, was unable to raise his head above his shoulders, manifested some lack of coördination of leg movement when walking and was dull and listless. He seemed better during the following summer but became worse again in the fall. Warm quarters, medicinal (salicylates) and dietary treatment did him no good.

On February 8, 1932, he was brought to the College clinic, where he was treated medicinally for two weeks with no beneficial results. Finally it was decided to treat him in the same way we had been doing with human patients in similar cases. Therefore, on February 25, 1932, cultures were made from his throat and feces. A streptococcus, showing a hemolytic zone with a greenish tint around the colonies on blood plates and showing weak fermenting properties, attacking dextrose only slightly and not the other common sugars, was isolated from the tonsils. *Escherichia coli* and another unidentified organism were isolated from the fecal sample.

Suspensions of killed cultures of these organisms (turbidity equal to that of tube 2 of McFarland's nephelometer) were injected intradermally in 0.1-cc doses into the lightest colored part of the skin in the anterior part of the inguinal region. In 24 hours, a rather distinct red area of the size of a quarter appeared where *E. coli* had been injected and a somewhat smaller area where the streptococcus had been injected. A mixed bacterial vaccine (bacterin) was therefore made of these two organisms and treatment was begun March 10, 1932. Six daily injections were made using a dose of 0.025 cc; then we made daily increases in the dosage of 0.01 cc.

On March 21, the dog showed improvement in his gait. The dose was increased daily until it reached 0.2 cc. From then on, injections were made at three-day intervals, increasing the dose every time by 0.02 cc. By the middle of April, the animal was definitely better in spite of the very cold and damp weather and the fact that he did not thrive very well on the hospital diet.

At the end of April, he seemed free from arthritis or rheumatism. He now held his head in a normal position, and could jump and move around otherwise in a normal manner. When brought to the Bacteriology Building, he ran up a very steep stairway without apparent effort and exhibited a decided preference for his caretaker. His attitude was decidedly different from that shown

when he was first examined, at which time he did not care as to who handled him. He was returned to his owner early in May. Some time afterwards he was killed by an automobile so we are unable to say how permanent his recovery was. The case of this dog was of particular interest because of the fact that the response was so much like that of many human patients treated in a similar manner for arthritis due to focal streptococcic infections.

This year, a seven-week-old doberman pincher (female) suffering from a similar ailment was apparently successfully treated with a hemolytic streptococcus vaccine, reinforced with a filtrate from the same streptococcus as that used in the vaccine. This pup had tonsillitis and showed symptoms suggesting that the joints in her legs were painful. She became tired very quickly when attempting to exercise and was rather dull and listless. According to the owner, this pup had shown leg weakness since birth. The carpal and phalangeal joints were enlarged. The grandmother and the mother of this pup had had difficulties in raising their pups, this pup being the only one surviving from a litter of three. Hemolytic and other streptococci were isolated from the vaginae of the grandmother and mother and from the throat of the pup. Treatment was started July 7, 1936, and was continued until July 31, 1936. The initial dose of vaccine was 0.05 cc. Injections were made intradermally in the flank every other day, the dose being increased by 0.02 cc each time for the first ten days of treatment. Later injections were made three or four days apart and the doses were increased by 0.03 cc each time. About 24 hours after the administration of the first dose, the pup was much worse and could hardly walk. She was also somewhat worse after each one of the next two treatments. From then on she improved steadily. By July 10, her tonsillitis had disappeared and at the end of the month she seemed well.

Discussion

The material presented here represents a number of clinical observations and the results of associated bacteriological and pathological examinations. We do not claim that we possess proof of the etiological importance of streptococci in early pup mortality. We believe, however, that, in view of our findings there seems to be far more evidence of infection being the cause of repeated loss of litters by some dams than that acid milk has anything to do with it. Milk samples examined by us showed no organisms other than what we concluded to be contaminants. *E. coli* was frequently isolated from pups as well as adults. We

also obtained a staphylococcus of the albus type on a number of occasions. *Staphylococcus aureus* was never found. It is too early to attempt to evaluate the rôle of the various organisms encountered, but the one streptococcus which was so frequently isolated from the female genital tract and which seemed to exhibit such a marked invasive power in pups, may be at least partly responsible for early pup mortality. Dr. Edwards,[11] after having examined four strains of this organism, wrote to the senior author in part as follows: "I have never encountered cultures having these reactions." It, therefore, seems that this is really a canine strain. For this reason we propose for it the name *Streptococcus canis N. S.*

It is, of course, possible that some of the other streptococci isolated may be pathogenic canine strains, but we have not had time to study them in sufficient detail to justify any attempts at classifying them. Further studies of the streptococci isolated from dogs, including pathogenicity determinations, are now in progress.

If infections are really responsible for early pup mortality, it means that one must employ appropriate sanitary precautions at whelping time and it may even be necessary to raise the pups apart from their mother.

The fact that many pups examined by us failed to show organisms of apparent etiological importance leaves room for much speculation and further work. Vitamin deficiency has been suggested as a cause of early pup mortality. In one kennel involved in these studies, the dogs were given vitamin supplements without the desired results.

SUMMARY

Evidence is presented in favor of infections rather than acid milk being responsible for early pup mortality.

A streptococcus commonly encountered in the female genital tract and pups that have died of so-called acid milk is described. The name *Streptococcus canis N. S.* is proposed for this organism.

Streptococci were found in pus from abscesses resulting from antirabies vaccination.

Two cases of articular and muscular rheumatism or arthritis, apparently successfully treated with vaccine therapy, are described.

ACKNOWLEDGMENT

We wish to thank Dr. P. R. Edwards, of the University of Kentucky, for checking some of our cultures.

REFERENCES

[1]Broadhurst, J.: Jour. Inf. Dis., xvii (1915), p. 277.
[2]Holman: Jour. Med. Res., xxxiv (1916), p. 377.
[3]Edwards, P. R.: Jour. Bact., xxviii (1932), p. 259.
[4]Edwards, P. R.: Jour. Bact., xxv (1933), p. 527.
[5]Lancefield, R. C.: Jour. Exp. Med., lvii (1933), p. 571.
[6]Plummer, H.: Jour. Bact., xxvii (1934), p. 465.
[7]Edwards, P. R.: Ky. Agr. Exp. Sta. Bul. 356 (1935).
[8]Zinsser, H., and Bayne-Jones, S.: Textbook of Bacteriology. (Appleton Century, New York, 1934), p. 330.
[9]Rice, T.: (W. B. Saunders Co., Philadelphia, 1935), p. 147.
[10]Devereux, E. D.: Amer. Jour. Pub. Health, xii (1932).
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DISCUSSION

DR. D. A. EASTMAN: I know so little about bacteriology that I feel rather embarrassed even to attempt to discuss a paper of this kind. I urge you to read it in its complete form. I believe there is information contained in it which we have never had before, and it is of value to all of us. However, I do wish to stress a few points.

The first, of course, is the importance of the possible answer to that portion of our pup mortality that breeders from time immemorial have referred to as being produced by acid milk, a diagnosis which most of us for a long time have not taken too seriously. On the other hand, when we were asked what the condition was, we felt it was very difficult to answer accurately.

The findings here, indicating the possibility of streptococcic infection as the cause of this baffling group of cases in new-born puppies, may save us a lot of embarrassment, and I urge you all to read this very well prepared paper and see what can be gleaned from it. I believe we have a possible answer to what has been a very embarrassing problem.

Dr. Stafseth also mentions the occurrence of streptococcic abscesses in the case report of an abscess following antirabic vaccination. I could not help but think how often I have criticized myself very bitterly when, after the delivery of a litter of puppies, I found myself confronted with a nice, big, typical streptococcic, not staphylococcic, abscess, following and resulting from the injections of pituitary extract. I am quite sure many of you have experienced that same thing. We knew the instruments were sterile. We had every reason to believe the pituitary extract was sterile and here we were confronted with these miserable abscesses. Again, perhaps the doctor has offered us a solution. I sincerely hope so. With a little more care taken to avoid contamination of the skin or our hands or any of the surrounding tissue with streptococcic infection of vaginal origin, we may be able to avoid many of these abscesses.

I wish to add one thing hurriedly in verification of the doctor's opinion that we do have a typically canine strain of streptococcus. These are just a few brief extracts from a report that was printed in the proceedings of the Society for Experimental Biology and Medicine, in 1936, by Dean D. J. Davis, of the College of Medicine of the University of Illinois, and his associates in this work. I, fortunately, happened to have some close contacts with it. I shall skim through some of the parts that might interest you.

"A study was undertaken to determine the relation of the hemolytic streptococci to this tonsillar infection and the incidence of similar streptococci in normal tonsils of dogs. All of the observations were made during the months of February, March and April, 1936.

"Cultures of fourteen pairs of tonsils yielded beta hemolytic streptococci in predominant number in nine instances, in pure culture in three. Of the five negative, three were overgrown with numerous colonies of the colon group. Three of the four swabs submitted yielded similar hemolytic streptococci, in one instance in pure culture. These

results indicate a high incidence of beta hemolytic streptococci, often in predominant or pure numbers in cultures from acutely inflamed tonsils.

"Five dogs were inoculated with a fresh strain of an encapsulated hemolytic streptococci (*S. epidemicus*) of human origin from a case of acute tonsillitis. The results were uniformly negative and at no time were the encapsulated mucoid streptococci recovered from the throat of the dogs.

"This same group of five dogs was inoculated in a similar way with a strain isolated from the lung of a second dog who died of distemper. Two became ill and revealed enlarged red tonsils, one with exudate, yielding large numbers of streptococci. Their temperature rose to 103.2° F. and 103.4° F. Recovery was apparent on the eighth day.

"Conclusions: (1) Hemolytic streptococci are constantly present in the tonsils of apparently normal dogs (92.3%). They occur in predominant numbers in acute tonsillitis of dogs and are secondary invaders in the bronchopneumonia of distemper. (2) Experimentally acute tonsillitis is produced by swabbing freshly isolated canine strains on the surface of the tonsils of dogs. A human strain (*S. epidemicus*) failed to produce a reaction. (3) The streptococci from dogs conform to the animal type and differ from the human type."

I think that is about all I have to offer, except to urge you again to take very seriously the work Dr. Stafseth has done on this and the practical advantage there may be in it to every practitioner.

Bruce Boyce Scores Again

Again, the son of an American veterinarian has enhanced the prestige of all Americans by his performance in the world of song. On March 14, Bruce Boyce, only offspring of Dr. Walter Boyce, a B. A. I. virus-serum inspector, stationed at Superior, Neb., scored another triumph. In the annual rendition of Bach's Saint Matthew Passion, sung by the Bach choir in Queen's Hall, London, he, as one of the soloists, earned a well-merited tribute from the musical critics of the day. Of his ability the following is quoted from one of the leading English newspapers:

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REFERENCES

[1]Broadhurst, J.: Jour. Inf. Dis., xvii (1915), p. 277.
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POST-SPLENECTOMY ANEMIA IN A COW; WITH A NOTE ON THE EOSINOPHILE CURVE*

By A. H. CRAIGE, JR., *Philadelphia, Pa.*
School of Veterinary Medicine, University of Pennsylvania

The subject, a mature Holstein cow, in an average state of flesh, was splenectomized in an effort to fulminate a possible anaplasma infection. At no time, either before the operation or during the nine months that followed, were the organisms in question identified in the blood of this animal. In fact, with the exception of the blood changes to be discussed, there was no evidence of change from the normal from the time that the cow was first observed. She had been suspected, at the beginning, of being infected with anaplasmosis, on the basis of non-specific symptoms and a supposed opportunity for infection. On reviewing the entire case history at the end of the study, it was agreed that the early suspicion was scarcely justified.

It is to be regretted that more extensive blood studies were not conducted prior to the time of operation. A single examination must suffice for this period. When the operation was performed, the animal was evidently in good health, all evidence of the earlier symptoms having disappeared. At this time, Dr. M. A. Emmerson, of the hospital staff, removed the spleen through a laparotomy wound in the flank, under the general chloroform anesthesia; the spleen, weighing 3 pounds, after the escape of about 1½ pints of blood, appeared normal in every respect.

Recovery was prompt and without complication. On the following morning, the animal was found eating and ruminating in a contented attitude; the wound was dry and only slightly swollen. From that time on to the end of the period of examination, symptoms of a general disturbance that could be associated with the anesthesia or the surgery were not observed. The erythrocyte changes to be described are, therefore, attributed to the loss of the splenic influence. The leukocyte changes are regarded as independent of even this factor.

*Received for publication, March 25, 1937.

It may be noted in table I that, while the erythrocytes showed a definite curve during the months succeeding this operation, the leukocytes were apparently unaffected. Fluctuating between 10,000 and 15,000, with a single rise to 19,000 six weeks after the time of operation, the component forms also failed to demonstrate any variations which could be ascribed either to the operation or to the removal of the spleen. Obviously the eosinophile curve could scarcely be classed as such.

TABLE I—*Blood levels.*

DATE	RED COUNT (x 10^6)	HEMOGLOBIN (GM / 100 CC)	RED VOLUME (%)	TOTAL LEUKOCYTES	EOSINOPHILES
1933					
11–29	8.5	11.0	35	10,850	1,800
12– 5*					
12– 6	9.2	11.5			
12– 8	8.0	10.5	35	13,200	1,800
12–15	7.4	9.75	33	10,500	600
12–19	7.0		30		
1934					
1– 9	6.8	7.0	24	18,950	2,300
1–13	5.0		25	13,500	
1–20†	5.8	7.5	25	15,150	3,000
1–24	5.0	7.5	26	15,400	
2– 5		7.5	21		
2–26	7.2	9.5		10,450	1,200
3– 5†	6.4	9.0	29	11,300	2,800
3–10	7.2	9.0	34	11,300	1,200
3–22	7.6	8.5		11,600	
4– 7	6.7	8.5	28	11,450	1,000
4–12	7.3	8.0	28	12,850	900
4–24	6.6	8.5	28	11,450	900
5– 1	5.2	7.0	26	10,000	
5–10	6.9	8.0	32	10,900	
5–18	6.9	8.0		12,700	

*Operation.
†At these times the cow showed unmistakable evidence of estrum.

The erythrocyte changes were manifest in a gradual decline in count, volume percentage, and hemoglobin content of the blood, for two months following the time of the operation. This period was in turn succeeded by one of partial recovery and maintenance at a level definitely below the preliminary and early postoperative figures. In comparing the graphic records of these curves, it is further evident that a partial relapse occurred later in the course of the reaction, having its peak about five months after the time of operation, and about three months behind the preceding comparable stage. It may be remarked in this connection that, although the subject presented much of the general appearance of

a normal cow, there were distinct symptoms of anemia coincident with the lower erythrocyte levels.

The circumstances surrounding this case make it one of considerable interest from the standpoint of physiology. While the reader would hesitate to draw any general conclusion from the results of a single case, and particularly where so few preliminary data are available, it is possible to compare these findings with those in other species and to show to what extent they agree. As to the influence of splenectomy upon the erythrocytes, opinion is divided: (1) That the spleen normally possesses an inhibiting influence upon erythrocytopoiesis, and removal is followed by accelerated elaboration of corpuscles with rise in count. (2) The larger group, that the splenic action in this regard is one of stimulation to the process of erythrocyte generation. Numerous cases of post-splenectomy anemia in man and the laboratory animals have been cited in support of the latter opinion.[1] The case in point serves to illustrate in the bovine species the same erythrocyte changes.

Following the white and differential counts over the same period, the eosinophile curve was noted to follow the fluctuations in the sexual cycle. A gradual rise to the peak, concurring with estrum and followed by a gradual return to the low interestrual minimum, was observed over two instances of the maximal eosinophile count. At these times, the levels were definitely outside the established normal range for the species (180 to 1,800 per cmm). In the intervals, the counts fell well within that range. The levels for neutrophiles, lymphocytes, basophiles and monocytes were uniformly within their respective normal ranges, and while they all showed some fluctuations, these were inconstant and not regarded as significant.

REFERENCE

[1]Krumbhaar: The changes produced in the blood picture by removal of the normal mammalian spleen. Amer. Jour. Med. Sci., clxxxiv (1932), p. 215.

A NEW LIGATURE FOR USE IN DEHORNING*

By JOHN F. RYFF, *Caledonia, Mich.*

After trying numerous methods in attempts to control the hemorrhage subsequent to dehorning cattle, I have found one which is an improvement over the use of twine. While I have heard of inner-tube elastics having been used before, they have never been employed in this particular fashion. Possibly this idea is not new to other veterinarians, however.

*Received for publication, May 4, 1937.

A wide elastic, made from an inner tube, is evenly looped double about the base of the horns before removal, much in the fashion of the usual cord. It is important that the band be placed well below the horn base in order to compress the soft structures. I carry an assortment of ½, ¾, and one-inch widths, using the wider bands where large horns are removed and the attendant danger of hemorrhage is greater. They are removed after the same interval, as with twine.

The advantages are that a constant tension is secured, no further adjustment is necessary, the band does not readily break, and it will not cut through the skin as wire might. While perhaps not so scientific as dissection and ligation of the artery, and also admitting of readily being copied by the farmer who does his own dehorning, the bands are quickly applied and their cost is negligible.

FURTHER RECORDS OF THE GIZZARD WORM, AMIDOSTOMUM ANSERIS, IN THE STATE OF WASHINGTON

Report of Cases in Wild Waterfowl*†

By A. C. JERSTAD, *Beltsville, Md.*

Zoölogical Division, U. S. Bureau of Animal Industry‡

Since a previous report[1] of the occurrence in the state of Washington of the gizzard worm, *Amidostomum anseris*, in domestic geese, the writer has observed this parasite in certain species of wild waterfowl, one of which, *Mareca americana*, appears to constitute a new host record. Three other cases constitute new locality records for *A. anseris* in the particular hosts (*Branta* spp.).

MATERIAL EXAMINED

Through the courtesy of Dr. F. W. Beckman and Dr. J. M. Smith, of the U. S. Bureau of Animal Industry, Olympia, Washington, and Mr. Theo. M. Sheffer, of the U. S. Bureau of Biological Survey, Puyallup, Washington, five specimens of gizzards were obtained from wild waterfowl taken under special permit at Long Beach, Washington, during January, 1936. The findings are summarized in table I.

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†This work was performed when the writer was with the U. S. Bureau of Animal Industry, at Puyallup, Wash., in coöperation with the Veterinary Department, Western Washington Experiment Station.

‡Assigned to this division by the Food and Drug Administration, U. S. Department of Agriculture.

TABLE I—*Results of the examination of gizzards.*

CASE	PARASITES	HOST*
1–W	12	*Branta canadensis canadensis* (Common Canada goose)
2–W	3	*Branta canadensis canadensis*
3–W	None	*Mareca americana* (American widgeon)
4–W	Few	*Branta canadensis leucopareia* (Lesser Canada goose)
5–W	2	*Mareca americana*

*Identified by Theo. M. Sheffer.

In each of the infested gizzards moderate gross lesions of amidostomiasis (erosion of the keratinoid layer with discoloration of the eroded and adjacent tissue) were noted. The Canada geese in cases 1-W and 2-W were reported to be in good condition. The widgeon in case 2-W, the only case in which no parasites were found, was thin. The remaining cases were declared to be in "medium condition."

DISCUSSION

Although only a limited number of gizzards were examined, these were taken entirely at random on two separate hunting trips; four out of five (80 per cent) were infested. Verbal reports by the hunters indicate that these birds were not so severely affected by this parasite as were some of the domestic geese in cases previously reported,[1] because they were in much better condition and were by no means stragglers from the flock. The number of specimens of *A. anseris* present in these birds, however, was far less than in the fatal cases referred to above.[1]

REFERENCE

[1]Jerstad, A. C.: The gizzard worm, *Amidostomum anseris*, of geese in western Washington. Jour. A. V. M. A., lxxxix (1936), n. s. 42 (3), pp. 318-320.

MIDWINTER EQUINE ENCEPHALITIS*

By L. P. DOYLE, *Lafayette, Ind.*

Purdue University Agricultural Experiment Station

During the past winter, there have occurred several, usually sporadic, cases of encephalopathy in horses. The impression exists among veterinarians and farmers that the cases this year have occurred mostly on farms where horses were not affected by a more general outbreak which occurred two years ago. In agreement with past experience, the cases this year have not been limited to horses having access to any particular kind of feed or pasture.

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The case reported here showed the first observed symptoms on January 14. There were eleven horses on this farm, and two of them became affected during a period of five days. The first horse to be affected died 15 hours and the second one died 12 hours after showing the first observed symptoms. Both animals walked in circles to the right and both developed violent nervous symptoms. One crashed through a fence, while the other one broke part way through the side of the barn.

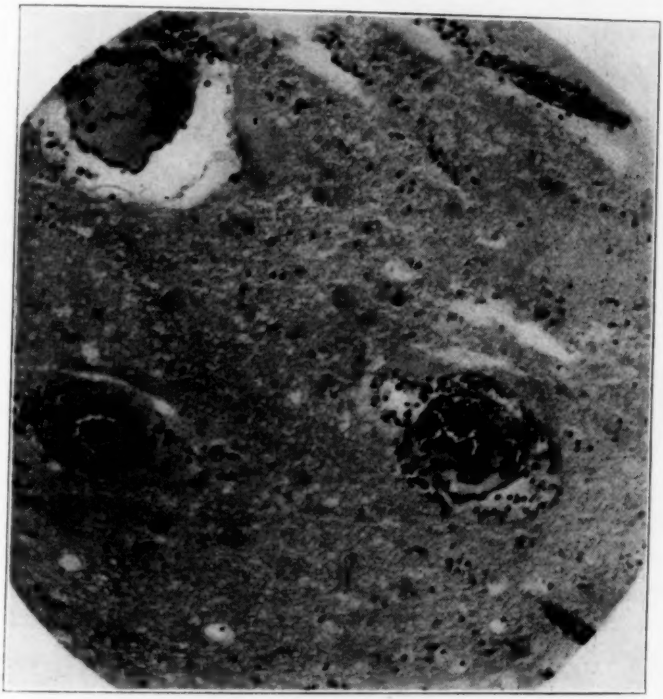

FIG. 1. A photomicrograph of a section of the brain, showing edema, peripheral and perivascular accumulations of cells, and degenerative changes in the brain substance.

The head of the second horse was brought to the laboratory for examination. Grossly, the brain showed marked congestion, edema and hemorrhages. There were two softened areas in the right cerebral hemisphere. These softened areas were yellowish in color; and one was about 1½ inches in diameter while the other one was smaller.

Microscopic examination of different portions of the brain showed that the major portion of the pathologic change occurred

in the cerebrum. In addition to the congestion, hemorrhage, edema and degenerative change apparent on gross examination, there were marked peripheral and perivascular accumulations of cells in and around the blood-vessels. These cells were mainly lymphoid in type, together with a good many eosinophils. The cell accumulations were found principally in the peripheral por-

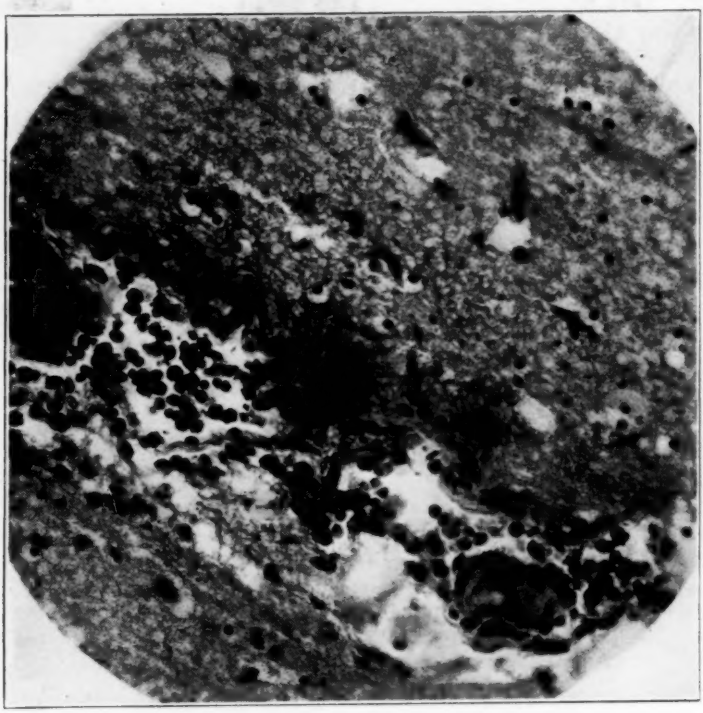

FIG. 2. A higher magnification of a section of the brain, showing the type of cell present.

tions of the softened areas, although they also occurred elsewhere. No cell inclusions were recognized in sections stained in hematoxylin and eosin.

The type of microscopic change found in this case is similar to what had been noted previously in cases occurring in the summer or fall. The vascular cuffing and encephalomalacia have been observed to occur together in previous cases studied. This is a point of considerable interest in connection with the question of whether one or more diseases are involved in the commonly occurring encephalopathy in horses. The opinion that the infectious type of disease is limited to the warmer portions of the year may not be so fully warranted as it is sometimes thought to be.

A Review of Certain Items in the Pharmacopoeia of the United States, Eleventh Revision, and the National Formulary, Sixth Revision, of Special Interest to Veterinarians

By H. D. BERGMAN, *Ames, Iowa*
Iowa State College

Perhaps before attempting the proposed brief review of the Eleventh Revision of the Pharmacopoeia of the United States and the Sixth Edition of The National Formulary, it might be well to recall the general significance of such publications and why they are essential to rational therapeutic practice. First, may it be said that most civilized countries have standards established by law to which many drugs, chemicals and pharmaceutical preparations, when sold or dispensed for medicinal use, must conform. The books in which these standards are described are ordinarily called Pharmacopoeias and the preparations listed therein are termed "official." In the United States there are two publications of this kind designated as legal standards, namely, the Pharmacopoeia of the United States (U.S.P.), first published in the year 1820 and revised decennially, and The National Formulary (N.F.), first published in the year 1888, and since 1896 revised decennially. The current revisions of these publications, designated U.S.P. XI and N.F. VI, superseded previous editions and became official standards on June 1, 1936. In the passage of the original federal Food and Drugs Act, June 30, 1906, by the United States Congress, the standards adopted under the Act were those of the U.S.P. and the N.F. The same standards hold for similar laws which have been enacted by certain of the states. The term "official" is just as properly applied to an N.F. product as to a U.S.P. product.

Certainly for accuracy, which means effectiveness and safety in therapy, some uniformity in the potency and preparation of medicinal products is absolutely essential. Within recent years, international standards have even been established for many of

the more potent drugs and pharmaceutical preparations, the basic principles of which have been adopted by most of the civilized countries. The first important conference aiming toward certain international standards for medicinal preparations was held in Brussels, Belgium, in September, 1902. It was called "The International Conference for the Unification of the Formulas for Potent Medicines" and was attended by delegates from nearly every civilized country. The purpose, as the name implies, was to attempt to formulate standards for *potent remedies* which would be adopted by the various pharmacopoeias of the world. A second conference, held in Brussels, in 1925, readopted most of the basic principles of the first conference. The U.S.P. has accepted many of these standards in the various revisions and has retained most of them. The abbreviation "P.I." (Protocol International), used in the United States Pharmacopoeia, designates those drugs and preparations for which basic uniform international standards have been adopted.

THE PHARMACOPOEIA OF THE UNITED STATES, ELEVENTH REVISION

The Pharmacopoeia is revised by a General Committee of Revision, elected by the United States Pharmacopoeial Convention. The latter consists of delegates from various incorporated pharmaceutical and medical organizations, including medical colleges, state and national associations, certain branches of the United States government, such as the Medical Departments of the Army and Navy, Public Health Service, and the Departments of Agriculture and Commerce.

In the U.S.P. XI, the general principles and policies of recent revisions have not been materially changed. The changes made have been largely to meet new requirements in revision methods and to take advantage of recent knowledge in related sciences in the development of tests for identity and purity, and assays for strength of the official medicinal substances. With the rapid advancements in the major scientific fields that have a bearing on therapy, especially in organic and biochemistry, the necessity for issuing more frequently the so-called "Interim Revisions" covering certain sections has become apparent, if the Pharmacopoeia and National Formulary are to represent the latest developments in therapeutic progress. A policy of issuing such revision supplements has been adopted and undoubtedly they will become increasingly important in maintaining pharmacopoeial standards representing the latest scientific developments. This is especially important because of the need of official standards in the enforcement of the Food and Drugs Act and by manufac-

turers of official medicinal products. Four "Interim Revision Announcements" were issued during the years 1934 and 1935. In the several proposals now before Congress concerning new federal Food and Drug legislation, the U.S.P. and N.F. are always recognized as the standards for the medicinal substances which they have admitted.

In the U.S.P. XI, the generally accepted structural formulas for organic chemicals have been added to the monographs, not as an indication of purity, but rather for the purpose of information. Important synonyms for the official articles are again included and substances designated by the official synonyms must meet the same requirements established for the substances under the official Latin names.

In order to establish greater uniformity in the preparation of percentage solutions in routine dispensing, the U.S.P. XI, following the example of the British Pharmacopoeia, has approved certain practical methods of procedure of interest to the practitioners. When the term "per cent" is used in dispensing, without qualifications, it is interpreted as follows: weight in volume for solutions of solids in liquids; volume in volume for solutions of liquids in liquids; and weight in volume for solutions of gases in liquids. For example, in the metric system, a 1 per cent solution is prepared by dissolving 1 gram of a solid or 1 cubic centimeter of a liquid in sufficient solvent to make 100 cubic centimeters of the solution, or, if prepared by the apothecaries system of weights and measures, by dissolving 4.5 grains of a solid or 4.8 minims of a liquid in sufficient solvent to make 1 fluidounce of solution. Also such phrases as "1 in 10," "1 in 100," etc., are interpreted to mean that 1 part by volume of a liquid, or 1 part by weight of a solid, is to be diluted with, or dissolved in, 10 or 100 parts by volume of the solvent.

While the metric system of weights and measures is the official system used in the new Pharmacopoeia, it is recognized in both the prescribing and dispensing of medicines that the old English apothecaries system of weights and measures is commonly used. In this connection, a point often overlooked is that in the apothecaries system, Roman, not Arabic, numerals are employed and these should always *follow* the symbol or abbreviation, as ℥v; gr. xx. In prescribing in the metric system, Arabic numerals are used and these should always *precede* the symbol, for example, 5 Gm.; 3 cc. Furthermore, in order to avoid the possibility of confusing the abbreviations for grain (gr.) and gram (Gm.),

with danger of serious error in dosage, the former is properly written with a small letter and the latter with a capital.

In referring to the metric system, the older members of the profession will recall the confusion that developed at the time of the adoption of the U.S.P. IX, on September 1, 1916, in which the term "cubic centimeter" (cc) was replaced by "mil," an abbreviation of the term "milliliter." One reason for the change was that the U. S. Bureau of Standards had pointed out that the cubic centimeter was not exactly the equivalent of a thousandth part of a liter. Another was that the term "mil" had been adopted by the British Pharmacopoeia. However, the change did not prove a popular one and was not generally accepted; hence, both U.S.P. X and XI returned to the use of cubic centimeter (cc). Actually, the trifling difference between the volumes of the cubic centimeter and the milliliter is negligible and of no significance in practical dispensing. Incidentally, the term "negligible," when used in the Pharmacopoeia, means a quantity not exceeding 0.0005 Gm.

As regards nomenclature in the new Pharmacopoeia, a notable change is that of the spelling of sulfur and derived titles such as sulfite, sulfate, thiosulfate, etc., in which "f" has replaced the former "ph." This change was recommended by the American Chemical Society and is in accordance with general practice. The N.F. VI has adopted similar spelling of these words.

Thirty-five changes were made in the titles of substances formerly official in the U.S.P. These were made for the purpose of insuring greater accuracy, brevity, or safety in dispensing or conformity with international usage. The changes in the official Latin titles that are of special interest to the veterinarian are listed below:

U. S. P. X	*U. S. P. XI*
Argenti Nitras Fusus	Argenti Nitras Induratus
Argento Proteinum Mite	Argentum Proteinicum Mite
Arseni Iodidum	Arseni Triiodidum
Caffeinae Sodio-Benzoas	Caffeina cum Sodii Benzoate
Chloramina	Chloramina-T
Dichloramina	Dichloramina-T
Ferri Hydroxidum cum Magnesii Oxido	Magma Ferri Hydroxidi
Hydrargyri Chloridum Corrosivum	Hydrargyri Bichloridum
Linimentum Saponis	Linimentum Camphorae et Saponis
Liquor Cresolis Compositus	Liquor Cresolis Saponatus
Liquor Hydrogenii Dioxidi	Liquor Hydrogenii Peroxidi
Liquor Pituitarii	Liquor Pituitarii Posterii
Oleum Cadinum	Pix Juniperi
Pulvis Glycyrrhizae Compositus	Pulvis Sennae Compositus
Sapo	Sapo Durus
Spiritus Glycerylis Nitratis	Spiritus Glycerylis Trinitratis

Fifty-eight new substances were deemed sufficiently important as therapeutic agents or pharmaceutical necessities to warrant pharmacopoeial adoption and 60 substances formerly official were omitted. Several of the latter have been included in the N.F. VI.

Among the 58 articles which were added to the U.S.P. XI, and have thus assumed official status, are several of special importance in veterinary therapeutics as follows:

Latin Title	*English Title*
Acriflavina	Acriflavine
Acriflavinae Hydrochloridum	Acriflavine Hydrochloride
Calcii Creosotas	Calcium Creosotate
Calcii Gluconas	Calcium Gluconate
Calcii Hydroxidum	Calcium Hydroxide
Carbo Activatus	Activated Charcoal
Chlorobutanol	Chlorobutanol
Emulsum Petrolati Liquidi	Emulsion of Liquid Petrolatum
Ephedrina	Ephedrine
Ephedrinae Hydrochloridum	Ephedrine Hydrochloride
Ephedrinae Sulfas	Ephedrine Sulfate
Liquor Ergosterolis Irradiati	Solution of Irradiated Ergosterol
Liquor Parathyroidei	Solution of Parathyroid
Oleum Maydis	Corn Oil
Phenacainae Hydrochloridum	Phenacaine Hydrochloride
Phenobarbitalum Solubile	Soluble Phenobarbital
Sodii Perboras	Sodium Perborate
Tinctura Iodi Mitis	Mild Tincture of Iodine
Tryparsamidum	Tryparsamide

The National Formulary, Sixth Edition

The National Formulary is revised by a "Committee on National Formulary" under the direct authority and supervision of the Council of the American Pharmaceutical Association. The Revision Committee of 15 members is assisted by numerous subcommittees and associate committees which have been appointed by various organizations having a special interest in medicinal products. In the National Formulary VI, veterinary medicine, for the first time, was given recognition in the revision of one of the legal drug standards through an invitation to the American Veterinary Medical Association to appoint a committee to represent the interests of veterinary medicine. Such a committee, known as the "Associate Committee on Veterinary Preparations," was appointed and coöperated in the revision. Incidentally, the A.V.M.A. has not been accorded membership in the United States Pharmacopoeial Convention, although the American Medical Association and the American Dental Association are each allowed three delegates.

The purpose of the N.F. is to furnish definite formulas for preparations extensively used in medical practice which are not included in the U.S.P. and to provide standards and tests for the identity, quality, and purity of the ingredients used in the

formulas, and for such other commonly used medicinal materials for which standards are not provided in the U.S.P. Formulas for preparations have steadily decreased in the U.S.P. and are now likewise decreasing in the N.F. Both are tending more to provide legal standards and tests for identity, assays, etc., of medicinal products, so that uniformity in therapeutic action may be assured.

One of the outstanding features of the N.F. VI is the admission of items on the basis of the definite extent of use in medical practice in the United States. The extent of use was determined by a nation-wide survey of hospitals, prescription pharmacies, drug-stores, etc., representing nearly every state in the Union. A study of the ingredients of thousands of prescriptions and medical formulas was made in widely separated states, and on the basis of such studies the tentative rule was adopted that items to be admitted to the N.F. VI must be used in at least 20 per cent of the pharmacies of the United States or must be an ingredient in at least one of each 10,000 prescriptions compounded. Only a few exceptions to this rule were made and these were for items classed as pharmaceutical necessities. An interesting result of the survey was the discovery that about two prescriptions per inhabitant are compounded annually in the pharmacies of the United States.

Another important feature of N.F. VI is the extensive development of the section on "Materials and Preparations for Diagnostic Use." Here are included instructions for the preparation of a wide variety of diagnostic reagents and clinical test solutions for the examination of urine, blood, gastric contents, spinal fluid, etc.; and also for the preparation of bacteriologic culture media and bacteriologic and histologic staining solutions.

In view of the extensive use in recent years of parenteral therapeutic methods which commonly involve the use of ampul preparations, the section in N.F. VI devoted to ampuls has been greatly enlarged. Under "Ampullae" (ampuls), specifications are described for the sterilizing of glass ampuls, preparation of ampul solutions, filling of ampuls, etc. Both single and multiple-dose ampuls are recognized, the latter being especially important in veterinary therapeutics. Twenty-eight ampul preparations are described in separate monographs; the strength of the ampul solution or suspension recognized in each monograph is the one most commonly used in medicine. Several of these preparations commonly used in veterinary medicine will be referred to later.

Reference was made previously to the A.V.M.A. committee appointed to represent the interests of veterinary medicine in the current revision of the National Formulary. While the ma-

jority of official drugs and preparations of the U.S.P. and N.F. have important uses in veterinary medicine, there are certain valuable medicinal products peculiar to veterinary therapeutics that are not important in human medicine and for which standards of purity, potency, etc., have not been established. An example is that of arecoline hydrobromide, the use of which is confined entirely to veterinary medicine and for which there has been no official standard with tests for identity and purity. The A.V.M.A. committee made a recommendation, which was accepted by the Revision Committee, for the inclusion of a limited number of such unstandardized drugs and preparations important in veterinary therapeutics. Therefore, in N.F. VI, there appear monographs including tests for identity, purity, assays, etc., and average veterinary dosage for such drugs as arecoline hydrobromide, areca, cambogia, kamala, potassium guaiacolsulfonate and tetrachlorethylene; also there are preparations such as a glycerinated-water solution of the alkaloids of nux vomica of a potency equivalent to that of the more expensive fluidextract of nux vomica and an ampul of calcium gluconate of 10 per cent concentration

Among the drugs and preparations that have been given official status by being added to N.F. VI are the following which are of special importance in veterinary therapeutics:

Latin Title	*English Title*
Ampullae Calcii Chloridi	Ampuls of Calcium Chloride
*Ampullae Calcii Gluconatis	Ampuls of Calcium Gluconate
Ampullae Dextrosi	Ampuls of Dextrose
Ampullae Dextrosi et Sodii Chloridi	Ampuls of Dextrose and Sodium Chloride
Ampullae Ephedrinae Sulfatis	Ampuls of Ephedrine Sulfate
Ampullae Epinephrinae Hydrochloridi	Ampuls of Epinephrine Hydrochloride
Ampullae Magnesii Sulfatis	Ampuls of Magnesium Sulfate
Ampullae Pituitarii Posterioris	Ampuls of Posterior Pituitary
Ampullae Procainae Hydrochloridi	Ampuls of Procaine Hydrochloride
Ampullae Sodii Citratis	Ampuls of Sodium Citrate
Ampullae Sodii Thiosulfatis	Ampuls of Sodium Thiosulfate
*Areca	Areca
*Arecolinae Hydrobromidum	Arecoline Hydrobromide
Calx	Lime (Quicklime)
*Cambogia	Gamboge
Cerii Oxalas	Cerium Oxalate
Gambir	Gambir (Pale Catechu)
Guaiacolis Carbonas	Guaiacol Carbonate
Hydrargyri Iodidum Rubrum	Red Mercuric Iodide
*Kamala	Kamala
Krameria	**Krameria**
Linimentum Calcis	Lime Liniment

*Veterinary dosage is given.

Liquor Arseni et Hydrargyri Iodidorum	Solution of Arsenic and Mercuric Iodides (Donovan's Solution)
Liquor Dextrosi et Sodii Chloridi Isotonici	Isotonic Solution of Dextrose and Sodium Chloride
*Liquor Nucis Vomicae Alkaloidorum	Solution of Nux Vomica Alkaloids
Liquor Plumbi Subacetatis	Solution of Lead Subacetate (Goulard's Extract
Oleoresina Capsici	Oleoresin of Capsicum
Oleoresina Zingiberis	Oleoresin of Ginger
Oleum Tiglii	Croton Oil
Phenol Camphoratum	Camphorated Phenol
Pilocarpinae Hydrochloridum	Pilocarpine Hydrochloride
Pilulae Hydrargyri Chloridi Mitis Compositae	Compound Pills of Mild Mercurous Chloride
*Potassii Guaiacolsulfonas	Potassium Guaiacolsulfonate
Pulvis Rhei Compositus	Compound Powder of Rhubarb (Gregory's Powder)
Rubrum Scarlatinum	Scarlet Red
Salicinum	Salicin
*Tetrachlorethylenum	Tetrachlorethylene

In addition to the above, there have been added a number of standard extracts, fluidextracts, ointments, solutions, tablets, etc., which are important in veterinary medicine.

Reference was made to the fact that in order for the U.S.P. to be maintained on a basis of representing the latest developments in the related sciences, provision is made for the preparation of supplements to the Pharmacopoeia at any time the Committee of Revision may deem such action desirable. A similar policy is followed by the National Formulary, whereby supplements may be prepared by the Committee on National Formulary and published as a part of the current edition. The N.F. recognizes the priority of U.S.P. standards to the extent that in case the U.S.P. in its supplements should approve items for admission already in the N.F. and establish standards for them, the Pharmacopoeial standards shall supersede those of the N.F. when official notice by publication has been made.

How a City Editor Describes a Cow

"The cow is a female quadruped with an alto voice and a countenance in which there is no guile. She collaborates with the pump in the production of a liquid called milk, provides the filler for hash, and at last is skinned by those she has benefited, as mortals commonly are."

Certified Milk.

THE EFFECT OF DIRECT ANIMAL PASSAGE OF THE PATHOGENICITY OF ENDOMOEBA HISTOLYTICA FOR KITTENS. Henry E. Meleny and William W. Frye. Amer. Jour. Hyg., xxv (1937), p. 313.

Two strains of *Endomoeba histolytica*, one showing a high and the other showing a low degree of pathogenicity in kittens, were each passed through seven dogs by direct transfer and were then returned to artificial cultivation. Each strain had previously maintained a relatively constant level of pathogenic activity to kittens over a period of three years of cultivation. Both strains became progressively more active in dogs by direct dog-to-dog transfer. After the seventh transfer, kittens were inoculated with material removed directly from infected dogs; the pathogenic activity of each strain was found to be markedly increased. After an interval of seven months in artificial cultivation, the strains were again tested in kittens. In the first test the pathogenic activity was lower than in the dog-to-kitten inoculation. When virulent hemolytic streptococci were added to the washed amebae of the less pathogenic strain and inoculated into kittens, there was no increase in the pathogenic activity of the amebae. Strains of *E. histolytica* from different sources may differ considerably in pathogenic activity. The increase in pathogenic activity which results from direct animal passage is due to a more favorable medium for the metabolic activity of the amebae.

STUDIES ON OÖCYST PRODUCTION IN AVIAN COCCIDIOSIS. I. A dilution count technique. Donald C. Boughton. Amer. Jour. Hyg., xxv (1937), p. 187.

A technic for counting oöcysts of avian coccidia by which dilution counts on suspensions of dried feces are made is described. Single counts readily differentiate the various levels of oöcyst production encountered in natural and experimental infections. Dried samples can be stored conveniently and do not deteriorate. The counting technic is capable of demonstrating parasite populations (*Isospora* in sparrows) of a markedly lower

order than those which can be observed microscopically in avian malarias.

STUDIES ON OÖCYST PRODUCTION IN AVIAN COCCIDIOSIS. II. Chronic isosporan infections in the sparrow. Donald C. Boughton. Amer. Jour. Hyg., xxv (1937), p. 203.

The oöcyst production of the coccidia (*Isospora*) of the English sparrow (*Passer domesticus*) under various caging conditions and as a result of experimental inoculation was studied by means of oöcyst counts. Infection is normally prolonged and chronic and is maintained for long periods in hosts which are not permitted access to infective material. An infection may readily be superimposed upon an existing chronic infection by inoculation of the host with infective material. A second experimentally induced infection can readily be established in a host which has previously experienced a similarly induced infection. The incubation period of a superimposed infection is approximately ten days. The time of day at which diurnal peak of oöcysts occurs is independent of the time of previous ingestion of infective oöcysts.

THE LOCALIZATION OF TRICHINELLA SPIRALIS IN THE MUSCLE OF ITS HOST. Charles H. Scheifley. Amer. Jour. Hyg., xxv (1937), p. 349.

Under the conditions of the experiment the diaphragms of four dogs were more heavily infested with *Trichinella spiralis* than the muscles of the extremities. Complete unilateral lumbar sympathectomy, with its resultant blood-flow, produced no significant change in the degree of localization in the operated limb as compared with the normal limb.

GUANIDINE RETENTION IN EXPERIMENTAL TRICHINOSIS OF RABBITS. Paul D. Harwood, Lloyd A. Spindler, S. X. Cross and Jessie T. Cutler. Amer. Jour. Hyg., xxv (1937), p. 362.

Guanidine intoxication is an important factor in many chronic diverse conditions involving lysis of large quantities of tissue or rapid fluid shifts. In five trichinosis-infected rabbits, concentrations of guanidine were found to be two to eight times that of normal rabbits. In one case tested, a very low blood sugar was found shortly before death from trichinosis. The enteritis of trichinosis seemed to be most severe in the duodenum, where the adult worms were least common; this inflammation may be par-

tially due to intoxication. The lesions in voluntary muscle caused by the larval trichinae probably gives rise to the guanidine found in the blood in trichinosis. Guanidine may be responsible for the acidosis found in the neighborhood of developing muscle trichinae. There seems to be little direct connection between trichinae and the nervous system and between the parasites and the pathology found in the brain, liver and kidney. In these points trichinosis and guanidinemia from other causes show parallel manifestations. Guanidine intoxication is regarded as causing the hemorrhages associated with trichinosis rather than the mechanical action of the migrating parasites.

THE ANTIHEMORRHAGIC VITAMIN. H. J. Almquist. Poultry Sci., xvi (1937), p. 166.

The results of various poultry nutrition experiments indicate the existence of a dietary disease in which chickens suffer from hemorrhagic tendencies and delayed blood-clotting. A review of the literature gives adequate support to the existence of an antihemorrhagic factor. The factor is stable to moderate heat. It can be isolated by extraction from dried alfalfa and is subject to concentration at least four fold. The hemorrhagic syndrome is characterized by a condition resembling scurvy, prolonged blood-clotting time, hemorrhages and anemia. External hemorrhages may develop suddenly with fatal conclusion. Exclusion of the antihemorrhagic vitamin has resulted in external hemorrhages in 75 per cent of the chicks within two weeks from hatching. Deficiency of the vitamin in commercial poultry feeds is extremely unlikely.

PULLORUM DISEASE IN DUCKLINGS. W. R. Hinshaw and H. A. Hoffman. Poultry Sci., xvi (1937), p. 189.

An outbreak of pullorum disease in week-old Mallard ducks is described. Pullorum disease was diagnosed in four-week-old Barred Plymouth Rock chicks from the same ranch simultaneously and evidence is advanced that the disease was transmitted to the ducklings from the chicks. The strains of *S. pullorum* isolated from the ducklings differed from laboratory strains. They differed in the production of a wrinkled growth on agar, formation of a slight pellicle on meat extract broth, and in their failure when first isolated to produce acid and gas in arabinose broth. After a year the duckling strain began producing acid and gas in arabinose medium. Reciprocal agglutination-absorption tests,

made with two tested laboratory strains as controls, showed the strains to be antigenically identical with *S. pullorum*. The duckling strains and chick strains produced typical pullorum disease in chicks but failed to infect one lot of two-day old Muscovy ducklings and one lot of eleven-day-old, Mallard-Pekin cross-bred ducklings.

SOME RESPONSES OF THE IMMATURE FEMALE FOWL TO INJECTIONS OF MARE GONADOTROPIC HORMONE AND ESTRIN. V. S. Asmundson, C. A. Gunn and A. A. Klose. Poultry Sci., xvi (1937), p. 194.

Young Leghorn and cross-bred Rhode Island Red and Plymouth Rock female chickens were injected for 5 to 42 days with pregnant-mare serum and/or estrin and killed when ten to 100 days old. The combs of Leghorns responded more quickly than those of the cross-breds although the latter had large turgid combs after prolonged injection with pregnant-mare serum. The ovaries of birds injected with pregnant-mare serum were enlarged. In the cross-bred birds injected for ten days and killed when 10 to 15 days old, there was an increase in interstitial tissue but no apparent effect upon the follicles. The ovaries of the older females, injected for five to 42 days and killed when 30 to 100 days old, were hyperemic, had increased interstitial tissue and enlarged follicles. No refractoriness was observed with increasing length of injection period but there was less response to a given amount of hormone in the older birds than in the younger birds. The oviduct was enlarged after injection with pregnant-mare serum or estrin. Prolonged injection (21 to 42 days) caused perforation of the vagina and secretion by the glands of the duct in the birds that were 42 to 63 days old at necropsy. The bursa of Fabricius was reduced in size after injection with estrin or pregnant-mare serum. The tibia tarsus was shorter in birds injected for 42 days with serum than in uninjected birds of the same age (63 days). The changes in the oviduct, bursa and long bones were presumably caused by the secretory activity of the ovary.

ALBINISM IN A COCKER SPANIEL. James McI. Phillips. Jour. Hered., xxviii (1937), p. 103.

The partial albinism in the cocker spaniel reported seems to be an example of that found in some Pekinese. Both the father and mother were red and white particolors. The litter consisted of six males, all red and whites with the exception of the case being

reported. The hair of the puppy at six months of age is pure white, eyes are blue, slight brown pigmentation occurs around the nose and foot-pads and the nails are colorless. Breeding tests will be conducted when the animal reaches maturity.

IMPACTED MOLARS—A NEW LETHAL GENE IN CATTLE. Edwin E. Heizer and Marshall C. Hervey. Jour. Hered., xxviii (1937), p. 123.

The occurrence of inherited lethal characters in cattle is reviewed and the condition of impacted molars, a lethal factor in milking Shorthorns, is shown to appear as if it were due to a single, simple recessive gene. Calves showing the deformity were of normal size but died during the first week after parturition. The animals showed an impaction of the premolar teeth in the mandible which was greatly reduced in length and width. The defective jaw was bilaterally symmetrical. The interalveolar space was longer than normal. The mandible was enlarged at the point of impaction. The incisor teeth were normal. Gross examination revealed no pathological conditions in other parts of the body.

LYSOZYME. The occurrence of an apparently sterile phase; the relation of lysis to dissociation. Max Pinner and Marie Voldrich. Jour. Inf. Dis., lx (1937), p. 7.

A bacillus is described that is particularly susceptible to lysozyme. Pleural effusions from tuberculosis patients contain lysozyme in relatively high concentration. Under appropriate conditions, secondary growth appeared following lysis; but there is a definite interval between lysis and secondary growth during which bacilli cannot be cultivated on media suitable for their growth. During this apparently sterile phase, the bacilli exist apparently in a biologically altered state, probably in the form of large Gram-positive cocci. Unpigmented and rough dissociates were observed to arise from the original yellow and smooth strain. The latter occurred more frequently in the secondary growth following lysis than in routine transplants.

SURVIVAL OF OXYGEN AND WATER DEPRIVAL BY TUBERCLE BACILLI. Truman Squire Potter. Jour. Inf. Dis., lx (1937), p. 88.

It is generally understood that oxygen deprival quickly kills tubercle bacilli. It is true that oxygen deprival does destroy the

growth of tubercle bacilli under a number of environmental conditions from which the inference is widely drawn that life also is destroyed. The experiments reported show that tubercle bacilli survive a deprival of oxygen far more severe than they are apt to meet in nature. Another fallacy regarding tubercle bacilli is that they are readily destroyed by desiccation. Tubercle bacilli survived in these experiments when the desiccation was rapid as well as when the desiccation was accomplished slowly and at 34° C. The author concludes that the severest deprival of oxygen and water yet accomplished does not guarantee the death of the tubercle bacilli but that under such conditions the bacilli in substantial numbers retain their viability.

A Study of Some of the Properties of the Toxic Substances Produced by Salmonella Paratyphi A and B. Ruth E. Gordon and C. N. Stark. Jour. Inf. Dis., lx (1937), p. 238.

Tests on the composition of the medium, the original reaction of the medium, and the incubation temperatures showed that toxic substances were produced in every instance in which the number of cells reached approximately 500,000,000 per cc before declining. The maximum potency of the toxic material was obtained under conditions which caused the number of cells to reach one to two billions per cc during the first three or four days of incubation and then slowly decline. In the absence of a fermentable sugar, the final reaction of the medium tended to approach pH 8.8 irrespective of its original reaction. A medium containing glucose caused a more rapid growth of the organisms but the pH limits were reached sooner. Boiling of aged cultures of *Salmonella* or of filtrates of these aged cultures for one hour did not materially affect the toxicity. Autoclaving aged cultures of *Salmonella* at 121° C. for one hour slightly decreased their poisonous effect. Autoclaving filtrates of aged cultures at the same temperature and for the same length of time markedly lessened the toxicity. Storage of toxic filtrates at 10° C. for two months and at 20° C. for one month did not influence their toxicity. Storage at 30° C. and 40° C. for two weeks slightly decreased the toxicity.

Doggone

Nine days from the time John Kleeman, of Lemmon, S. Dak., gave his collie to friends who took it 200 miles to Ashley, N. Dak., the dog was back pawing with swollen feet at his master's door. The Kleemans were as glad to have the dog back as he was to get home. *Farm Journal.*

Regular Army

1st Lt. Lloyd C. Tekse is relieved from further assignment and duty at the Army Veterinary School, Army Medical Center, Washington, D. C., and from further temporary duty as a student, Medical Field Service School, Carlisle Barracks, Pa., effective in time to comply with this order, is then assigned to station at Fort Mason, Calif., and will proceed at the proper time to New York, N. Y., and sail on the transport scheduled to leave that port on or about August 4, 1937, for San Francisco, Calif.; upon arrival in San Francisco will report to the commanding general, San Francisco Port of Embarkation, for duty.

1st Lt. Velmer W. McGinnis is directed to proceed from Carlisle Barracks, Pa., to his proper station, Army Medical Center, Washington, D. C., effective upon completion of his present course of instruction at the Medical Field Service School, and upon arrival will report in person to the commanding officer, General Dispensary, U. S. Army, Washington, D. C., for temporary duty for approximately two months; upon completion to return to his proper station Army Medical Center.

Major Burlin C. Bridges is relieved from duty at Fort Ethan Allen, Vt., effective at such time as will enable him to proceed to New York, N. Y., and sail on or about August 4, 1937, via Government transportation, for the Hawaiian Department, for duty.

Lt. Col. Harold E. Egan is assigned to duty at Fort Francis E. Warren, Wyo., effective upon completion of his present tour of foreign service in the Hawaiian Department.

Major Joseph H. Dornblaser is relieved from his present assignment and duty at the Quartermaster Remount Depot, Fort Reno, Okla., effective on or about July 5, 1937, is then assigned to station at Fort Worth, Texas, will proceed to that station and report to the officer in charge, Remount Area Headquarters, for duty.

Veterinary Reserve Corps

NEW ACCEPTANCES

Emerson, Harry Howard	Capt.	6433 S. May St., Chicago, Ill.
Boyink, Gordon Darrow	1st Lt.	174 Gibbs St., Rochester, N. Y.
Fisher, Sam Sylvester	1st Lt.	R. 1, Port Austin, Mich.
Lawrence, Walter A.	1st Lt.	125 N. W. 23rd St., Oklahoma City, Okla.
Walters, Maurice King	1st Lt.	310 W. T. Waggoner Bldg., Fort Worth, Texas.
Weight, Leslie Askew	1st Lt.	Wailuku (Maui), T. H.

PROMOTIONS

To

Harlan, Wm. Henry	Capt.	1901 W. Park St., Oklahoma City, Okla.

Miller, Fred Henry .....Capt...East Side Market Sq., Wausau, Wis.
Fitzgerald, Theo. C. .....1st Lt...2710 Zollinger Rd., Columbus, Ohio.
Hayes, Raymond Wm. .....1st Lt...1379 N. Prairie St., Galesburg, Ill.
Hinchman, Howard R. ....1st Lt...Canal Winchester, Ohio.
Linzinmeir, Chas. B. .....1st Lt...124 S. Washington Ave., Columbus, Ohio.
McKenzie, Carl Van.....1st Lt...1174 W. 5th Ave., Columbus, Ohio.
McKitrick, John Leslie ....1st Lt...Station B, R.1, Columbus, Ohio.
Scothorn, Marion W. .....1st Lt...209 S. Maple, Newkirk, Okla.
Tanner, Warren Leslie ....1st Lt...304 E. Main St., Van Wert, Ohio.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Harden, Chester N. .....1st Lt...Hq. 1st Corps Area, Boston, Mass.
Willers, Karl H. ,.....1st Lt...Presidio of San Francisco, Calif.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Prchal, Charles Frank.....1st Lt...Fort Crook, Neb.

Radio Talks Urge Vaccination

A radio campaign over twelve midwestern stations, urging farmers to have their hogs vaccinated against cholera by a veterinarian, is being launched this month as a new development in the educational campaign sponsored by the Associated Serum Producers.

The radio effort will consist of spot announcements on farm programs in the early morning and during the noon hour, on an average of three to five days per week. The announcements will begin around weaning time and will continue through June.

Stations being used include: WLS, Chicago; KMBC, Kansas City; KFAB, Lincoln; WOW, Omaha; WBNS, Columbus, Ohio; WCCO, Minneapolis; KSOO, Sioux Falls, S. Dak.; WOWO, Fort Wayne, Ind.; WCBS, Springfield, Ill.; KRNT, Des Moines; WMT, Waterloo, Iowa; WHO, Des Moines; and KFNF, Shenandoah, Iowa.

The radio copy emphasizes the dangers of hog cholera, the wisdom of having spring pigs vaccinated around weaning time, and the importance of having the vaccinating done by a veterinarian.

The Associated Serum Producers have also announced plans for a new series of summer radio talks, to take up summer diseases of live stock and to be presented, starting in June or July, over leading college and commercial stations.

COMMENCEMENTS

ONTARIO VETERINARY COLLEGE

The following candidates received the degree of Bachelor of Veterinary Science at the Convocation of the University of Toronto, May 14, 1937:

S. D. Ahmad
R. McG. Archibald
A. O. Beemer
F. C. Bridgman
F. E. Burlingame
G. V. Burton
C. H. Chapman
J. A. Charlton
R. J. Cyrog
T. E. Darlington
F. J. Fitzgerald
J. A. Folinsbee
C. K. Francis
F. W. Gehrman
E. H. Hageman
H. H. Hamilton
J. R. Hasson
T. W. Hogarth
J. T. Horel
G. M. Johnson
F. M. Jones
J. M. Livingstone
R. S. MacDonald
E. C. Moore
R. D. F. Parr
N. H. Peter
R. J. Pinkney
W. F. Riley
A. L. Schaefer
W. G. Stevenson
C. L. Turner
D. D. Watson

Prizes were awarded to members of the graduating class as follows:

Honor Standing

Gold Medal for highest aggregate standing—W. F. Riley, of Watertown, Conn.
Second Prize—W. G. Stevenson, of Cobden, Ont.
Third Prize—J. A. Charlton, of Paris, Ont.

Andrew Smith Memorial Medal

W. F. Riley, of Watertown, Conn.

Helen Duncan McGilvray Prize

J. A. Charlton, of Paris, Ont.

Bacteriology

W. G. Stevenson, of Cobden, Ont.

Canadian Army Veterinary Corps Prize

R. J. Pinkney, of Cooksville, Ont.

Kansas State College Recognition Day

The fourteenth annual Recognition Day program of the Kansas State College took place in the College Auditorium, Manhattan, April 14, 1937. The names of the prize-winners in the Division of Veterinary Medicine are as follows:

Physiology

Prizes offered by Dr. N. D. Harwood (K. S. C. '18), of Manhattan, Kan.
First Prize ($10).....Norwood Harry Casselberry, '39
*Second Prize ($2.50).....Wade Oberlin Brinker, '39
*Second Prize ($2.50).....David E. Evans, '39

Therapeutics

Prizes offered by Dr. Chas. E. Salsbery (K. C. V. C. '11), of Kansas City, Mo.
First Prize ($10).....Joseph Sterling, '38
Second Prize ($5).....Sidney Levine, '38

Pathology

Prizes offered by Dr. O. M. Franklin (K. C. V. C. '12), of Amarillo, Texas
First Prize ($10).....Irwin John Twiehaus, '37
Second Prize ($5).....Willard M. Van Sant, '37

Small-Animal Medicine

Prizes offered by Dr. C. W. Bower (K. S. C. '18), of Topeka, Kan.
First Prize ($10).....Lee Thomas Railsback, '37
Second Prize ($5).....Clark Wayne Burch, '37

General Proficiency

Prizes offered by Drs. Benj. F. Pfister (K. S. C. '21) and Earl F. Hoover (K. S. C. '24), both of Kansas City, Mo.
First Prize ($15).....Edwin Morris Crawford, '37
Second Prize ($10).....Guy William Bayles, '37

Cornell University Honor Day

Honor Day exercises of the New York State Veterinary College, at Cornell University, Ithaca, were held May 11, 1937, with Dean W. A. Hagan presiding. Dr. Chas. M. Carpenter (Corn. '17), Director of Laboratories, Rochester Health Department, Strong Memorial Hospital, Rochester, N. Y., delivered the opening address and President Livingston Farrand awarded the prizes as follows:

Addison D. Merry Prize

Given by Dr. A. E. Merry (Corn. '06), Syracuse, N. Y., for the best work in veterinary anatomy.
First Prize ($30).....Wilson Bryan Bell, '39
Second Prize ($20).....John Delaney Murray, '39
Honorable Mention: Lyndon Wainwright Potter, '39.

Jane Miller Prize

Given by Dr. Frank H. Miller (Mont. V. C. '87), New York, for the best work in veterinary physiology.
*First Prize ($20).....Alexander Morris, '38
*First Prize ($20).....Robert B. Morris, '38
Honorable Mention: Stephen James Roberts, '38.

Charles Gross Bondy Prize

Given by Mr. Richard Bondy, New York, for the best work in the courses in practical medicine and surgery of the small animals.

First Prize ($25) .....Leon Jules Lacroix, '37
Second Prize ($15).....Elwood Stephen Francis, '37
Honorable Mention: George Conrad Schloemer, '37 and Morris Siegel, '37.

Anne Besse Prize

Given by Miss A. B. Jennings, New York, for the best work in medicine and clinical diagnosis.
First Prize ($25) .....Victor Frost Van Wagenen, '37
Second Prize ($15) .....Albert Louis All, '37
Honorable Mention: Alfred Francis Nolan, '37 and Irwin Herbert Roberts, '37.

James Gordon Bennett Prize

Given by James Gordon Bennett, New York, for the students who show the greatest humaneness in handling animals, with special reference to the use of anesthesia.
First Prize ($30) .....George Greenwood Pickett, '37
Second Prize ($20) .....Jack Edward Baker, '37
Honorable Mention: Elwood Stephen Francis, '37 and Leon Jules Lacroix, '37.

Horace K. White Prize

Given by Horace K. White, and his sons, of Syracuse, for the students whose academic records for the entire veterinary course stand highest.
First Prize ($75) .....Dorsey William Bruner, '37
Second Prize ($25) .....Arnold Eder, '37
Honorable Mention: Herbert Racoff, '37 and Irwin Herbert Roberts, '37.

SKYLINE OF OMAHA AS SEEN FROM THE UNION PACIFIC SHOPS

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The fifty-third annual meeting of the Veterinary Medical Association of New Jersey was held at the Hotel Douglas, Newark, on January 14-15, 1937. President E. R. Cushing, of Plainfield, presided at the two-day program which included a business session, literary program, Association dinner and a "question-box" feature. Following the business session on the first day, four excellent talks were given as follows:

"Calfhood Vaccination Against Bang's Disease," by Dr. L. J. Tompkins, New York, N. Y.

"Observations Made in Twenty Years of Immunological Work," by Dr. B. M. Lyon, Pearl River, N. Y.

"Some Newer Aspects of Animal Disease in Relation to Humans," by Dr. John Eiman, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.

"Show Dogs and Dog Shows," by Dr. C. J. McAnulty, Atlantic City, N. J.

At 7:00 p. m., the Association dinner was held, the Ladies Auxiliary being especially invited for this feature. The local committee deserves great credit for the splendid dinner and entertainment provided. One hundred twenty members, guests, and their ladies were present, music for dancing during dinner being furnished by a local orchestra. Following the dinner, Dr. Earle P. Perkins, of the Department of Biology, Rutgers University, showed his motion-picture films taken as a member of the scientific staff of the Second Byrd Antarctic Expedition. Dr. Perkins described life and experiences at "Little America" in a most instructive and entertaining way.

On the morning of January 15, the following excellent program was presented:

"Disease Control Problems in Commercial Dairy Herds," by Dr. R. S. Huff, Newton, N. J.

"Canine Practice and Nutrition," by Dr. W. J. Lentz, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.

"Bovine Mastitis and Its Control," by Dr. D. H. Udall, New York State Veterinary College, Cornell University, Ithaca, N. Y.

After lunch, Dr. E. E. Sweebe, of North Chicago, Ill., presented a motion-picture film and talked on the development and uses of the barbiturates in anesthesia.

Following a report of the Executive Committee and the election of officers, the rest of the afternoon was given over to a question-box session which developed a great deal of interest and discussion.

In recognition of the outstanding services of the late Dr. John H. McNeil, the Association took action to provide a suitably enlarged and framed photograph of Dr. McNeil for the New Jersey Bureau of Animal Industry.

An amendment of the Constitution and By-laws was proposed which would facilitate transfer of membership of veterinarians in the employ of the federal government. This amendment will be acted upon at the next meeting, as will also another one aimed to simplify the procedure of handling delinquent membership.

At the business session, the following officers were elected for the current year: President, C. J. McAnulty, Atlantic City; 1st vice-president, G. H. Kimnach, Hightstown; 2nd vice-president, P. F. Runyon, Freehold; treasurer, J. B. Engle, Summit, and secretary, J. G. Hardenbergh, Plainsboro.

Mention should be made of the excellent commercial exhibits, participated in by 15 firms, which have become a welcome and worth-while addition to the annual meeting.

J. G. HARDENBERGH, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The February meeting of the Association was held at the Hotel New Yorker on Thursday evening, February 25, 1937, with Dr. R. S. MacKellar, Jr. presiding.

Col. Robert J. Foster, president of the American Veterinary Medical Association, gave a very fine address concerning the importance of organization for veterinarians. He pointed out the fact that it is very essential for all veterinarians to be members of their local, state and national associations. He stated that in comparison with our sister professions, medicine and dentistry, the percentage of membership in our national association is very low. At the conclusion of his talk, Col. Foster gave several concrete recommendations for the betterment of the A. V. M. A.

Dr. J. R. Mohler, Chief of the U. S. Bureau of Animal Industry, gave a brief, but interesting, travelog of his recent trip to Europe.

Dr. W. A. Hagan, Dean of the New York State Veterinary College at Cornell University, gave a talk on veterinary organizations. He stressed the point that there should be close connections between local and state organizations. This would make one strong organization with a large membership which would be in a better position to protect the welfare of our profession.

Dr. O. E. McKim, of Port Chester, N. Y., made an earnest plea in the interest of our destitute fellow practitioners in the flooded area. As a result, $117.00 was raised.

J. B. ENGLE, *Secretary.*

CHICAGO VETERINARY MEDICAL ASSOCIATION

With the meeting of March 9, 1937, the Chicago Veterinary Medical Association transferred its regular meeting-place from the Palmer House to the Anti-Cruelty Society headquarters, located at 157 West Grand Avenue. Through the kindness of Dr. W. A. Young, Manager, and the Board of Directors of the Anti-Cruelty Society, spacious meeting rooms have been placed at the disposal of the Chicago veterinarians and their wives for the monthly meetings of the Association.

At the March meeting, Dr. Young gave a very interesting talk on animal welfare work as conducted by local and national humane organizations. He emphasized the importance of the veterinary phases of humane work with animals.

APRIL MEETING

At the meeting held April 13, 1937, Dr. D. M. Campbell, chairman of the A. V. M. A. Committee on Public Relations, and Dr. H. Preston Hoskins, chairman of a similar committee of the Chicago Veterinary Medical Association, discussed the activities which could properly be included in a program for a committee on public relations.

MAY MEETING

The last regular meeting of the season was held May 11, 1937. Drs. E. C. Khuen, of Evanston, and G. S. Elwood, of Chicago, gave comprehensive reports on the meeting of the American Animal Hospital Association which was held in Detroit, Mich., May 6-8. Senate Bill 2312, which would provide a brigadier-

general for the Army Veterinary Corps, was discussed. All members were asked to write their congressmen urging support of this bill.

EAST CENTRAL (IOWA) VETERINARY ASSOCIATION

A dinner-meeting of the East Central Veterinary Association, held at the Hotel Montrose, Cedar Rapids, Iowa, April 8, 1937, was attended by 47 practitioners from 17 counties. Dr. Samuel S. Paul, of Clarence, presided.

Dr. C. H. Covault, of Iowa State College, Ames, gave an interesting lecture on "Equine Influenza." He outlined excellent treatments for the animal already infected but pointed out the limitations of the present-day preventive treatments. An interesting discussion followed. Dr. F. J. Crow, of Iowa City, presented an interesting case report on impaction in baby chicks due to a lack of grit in commercial feed. Diseases of baby chicks were discussed by several veterinarians.

Dr. V. G. Bromwell, of Center Point, was unable to be present, so his paper on "Handling Diseases of Foals," was read by the secretary, Dr. Edw. H. Beretta, of Solon. Dr. R. E. Elson, of Vinton, gave an interesting paper on "Calf Diseases." Dr. Iva Dunn, of Atkins, gave a paper on "The Handling of Diseases of Suckling Pigs."

The guest speakers included Dr. H. A. Seidell, Chief of the Iowa Bureau of Animal Industry, Des Moines; Dr. J. A. Barger, Inspector-in-Chief, U. S. Bureau of Animal Industry, Des Moines, and Dr. C. E. Hunt, president of the Eastern Iowa Veterinary Association, Mount Pleasant.

FAYETTE COUNTY (IOWA) VETERINARY MEDICAL ASSOCIATION

Twenty-six veterinarians from eleven counties attended the monthly meeting of the Fayette County Veterinary Medical Association held at the Hotel West, West Union, Iowa, April 20, 1937. Dr. C. L. Crider, of Elkader, presided at the meeting.

Dr. P. V. Neuzil, of Blairstown, was the principal speaker on the program. His subject was "Baby Chick Diseases, Their Control and Prevention." Dr. Neuzil urged each practitioner to make better use of his opportunities to serve poultry-raisers. He stated that disease is a great menace to profitable poultry-raising and none are better qualified to understand and control poultry diseases than are veterinarians in practice. He pointed out the great danger from leukosis to the poultry industry

through the use of eggs from infected hens used for hatching purposes. Dr. Neuzil pointed out the success attained by turkey-raisers in the elimination of disease by sanitation. He believes that chicken-raisers will be compelled to follow the example set by turkey-raisers.

Dr. H. A. Seidell, Chief of the Iowa Bureau of Animal Industry, Des Moines, spoke on "Sheep Scabies and Its Control." Dr. M. L. Spear, of Oelwein, gave a number of case reports covering his efforts, through the use of biologics, to prevent equine influenza in farm horses transported by rail. Dr. M. F. Frever, of West Union, presented a case report on recurrent exuberant granulations in a horse. Discussions were given by Drs. C. L. Crider, N. A. Kippen, of Independence, and H. H. Thomson, of Postville.

SOUTHWESTERN MINNESOTA VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Southwestern Minnesota Veterinary Medical Association was held in the Legion Hall, Truman, Minn., April 27, 1937.

Prof. W. E. Peterson, University Farm, Saint Paul, Minn., presented a paper on "'Feeds and Feeding." He emphasized the problem of utilizing feeds which are deficient in minerals and vitamins. Dr. Frank Breed, Lincoln, Neb., presented a paper on "Diseases of Sheep and Milk Fever in Cattle."

All veterinarians in the Veterinary Division at the University of Minnesota were made honorary members of the Association. A banquet was served following the meeting, with 60 veterinarians in attendance.

The following officers were elected for the ensuing year: President, Dr. C. J. Gillespie, Heron Lake; vice-president, Dr. T. F. Bartley, Vesta; secretary-treasurer, Dr. L. E. Stanton, Jackson, and member of the Board of Trustees, Dr. J. A. Roberts, Saint James.

L. E. STANTON, *Secretary.*

NORTH CENTRAL IOWA VETERINARY MEDICAL ASSOCIATION

The North Central Iowa Veterinary Medical Association held a meeting at the Hotel Warden, Fort Dodge, April 28, 1937, with Dr. J. O. F. Price, of Algona, presiding. Approximately 200 veterinarians were in attendance.

The meeting was opened with an address by the president. Dr. L. D. Frederick, of Chicago, Ill., read a paper on sheep diseases in which special attention was directed to pregnancy disease. In addition, he referred to the feeding of sheep. Dr. H. E. Schwermann, of New Ulm, Minn., discussed "Selling Your Services." A case report on sweet clover poisoning was presented by Dr. F. E. Williams, of Duncombe. The discussion was given by Drs. H. J. Shore, of Fort Dodge, J. R. Roche, of Gowrie, and F. B. Young, of Waukee. Interesting case reports of pioneer veterinary practice were related by Drs. W. L. Evers, of Iowa Falls, N. A. Kippen, of Independence, and D. E. Baughman, of Fort Dodge.

Experiences encountered in recently established practices of veterinarians were given by Drs. M. W. Osburn, of Independence, R. L. Wiedemeier, of Lytton, and J. D. Shoeman, of Dallas Center. Dr. J. C. Carey, of West Liberty, president of the Iowa Veterinary Medical Association, addressed the members. Dr. H. A. Seidell, Chief of the Iowa Bureau of Animal Industry, Des Moines, made comments concerning some state regulations. Dr. J. A. Barger, of Des Moines, Inspector-in-Charge, U. S. Bureau of Animal Industry, discussed Bang's disease.

The visiting ladies were entertained at a luncheon which was followed by a theatre party. In the evening, the veterinarians and their wives attended a banquet and dance. The entertainment included motion pictures (colored) of Mexico, presented by Dr. F. L. Knowles, an orthopedic surgeon of Fort Dodge.

The election of officers resulted as follows: President, Dr. P. O. Dorweiler, West Bend; vice-president, Dr. R. C. Ball, Titonka, and secretary-treasurer, Dr. H. C. Smith, Fort Dodge.

H. C. SMITH, *Secretary.*

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Michigan-Ohio Veterinary Medical Association was held in Blissfield, Mich., on May 21, 1937, with Dr. J. W. Marshall, of Genoa, Ohio, presiding.

The clinic which had been arranged was not fully carried out because of the heavy downpour of rain which prevented outdoor work. After a dinner at the Masonic Temple, the literary program was given.

Dr. C. W. Witty, of Elmore, Ohio, presented a paper on "Calcium Therapy." Dr. W. R. Hansen, of Shelby, Ohio, dis-

cussed a large number of cases which he had treated during the past few months. Dr. E. C. W. Schubel, of Chicago, pointed out the benefits of organized veterinary medicine and the advantages to be obtained through membership in the American Veterinary Medical Association. Dr. Schubel tendered his resignation as secretary-treasurer after having served the organization for eight years.

The election of officers resulted as follows: President, Dr. F. E. Caswell, of North Adams, Mich.; vice-president, Dr. W. R. Hansen, of Shelby, Ohio, and secretary-treasurer, Dr. J. C. Schwabland, of Blissfield, Mich.

J. C. SCHWABLAND, *Secretary.*

Eastern States Veterinary Conference

The keen interest taken by the eastern states in pooling their resources in arranging for the combined meeting of the veterinary associations of New York, Pennsylvania, New Jersey, Maryland, Delaware, the District of Columbia and the New England states assures a good meeting.

The meeting will be run in three sections. There will be a large-animal section, a small-animal section and a section on meat and milk hygiene. Poultry, pet birds and educational exhibits will receive attention.

The whole meeting, including the clinic, is to be held in different ballrooms on one floor of the Hotel New Yorker, New York City, which is air-cooled and especially adapted to a meeting held during warm weather. The dates: July 7-8-9.

All veterinarians welcome!

ORGANIZING COMMITTEE,
C. P. ZEPP, *Chairman.*

Magnesium Sulfate as an Insecticide

Grasshopper hordes are forecast for the coming summer in the Midwest. The usual method of poisoning is by the use of arsenic, which, unfortunately is poisonous also to live stock. Recently it was reported in *Science* that magnesium sulfate could be used as an insecticide. Grasshoppers have been destroyed as effectively with magnesium sulfate as with a 5 per cent arsenic bait. Magnesium sulfate is cheaper and it appears to be practically harmless to humans, cattle, swine and poultry. It may be used safely as a spray on many vegetables and fruits, with little danger to humans and animals eating such foods.

NECROLOGY

CLARENCE A. TOWNE

Dr. Clarence A. Towne, of McGraw, N. Y., died at the home of his mother, in Cortland, N. Y., on February 22, 1937, at the age of 51 years. He was a graduate of New York State Veterinary College at Cornell University, class of 1907. He practiced first at Oneonta, N. Y., later being associated with the S. P. C. A. of Syracuse. He had practiced at McGraw for about four years.

WILLIAM C. MILLER

Dr. William C. Miller, of Long Beach, N. Y., died in the Misericordia Hospital, New York City, on March 14, 1937, following a brief illness.

Born in New York City, August 28, 1880, Dr. Miller was a graduate of the New York-American Veterinary College, class of 1904. For a number of years he was Chief Veterinarian to the New York City Police Department. Then he became Chief Veterinarian to the Health Department, a post he held for 23 years.

Dr. Miller joined the A. V. M. A. in 1929. He is survived by his widow (née Elizabeth F. Murphy), two sons and one daughter.

JOSEPH C. PHILLIPS

Dr. Joseph C. Phillips, of Lethbridge, Alberta, Can., died suddenly on March 26, 1937, from perforated gastric ulcers.

Born in Hampsthwaite, Yorkshire, England, June 10, 1878, Dr. Phillips located at Estevan, Saskatchewan, in 1901. He started his veterinary studies at the Chicago Veterinary College, where he spent two years. He then transferred to the Ontario Veterinary College and was graduated with the class of 1921. He practiced in eastern Canada for a short time and then entered the service of the Health of Animals Branch, Canada Depart-

ment of Agriculture. He was first stationed at Edmonton and then at the Veterinary Research Station at Lethbridge.

Dr. Phillips joined the A. V. M. A. in 1923. He is survived by his widow (née Sarah Clark), five sons, one brother and two sisters. Two of the sons are veterinarians, Drs. N. C. Phillips (Ont. '32), and F. C. Phillips (Ont. '34), both of Edmonton, Alta.

L. M. H.

RAUS P. FRANS

Dr. Raus P. Frans, of Monmouth, Ill., died at his home, April 10, 1937, after a lingering illness dating back to December last. Owing to failing health, he gave up active practice about five years ago and since that time had been the local representative of the Equitable Life Assurance Society of the United States.

Educated in the public schools of Henderson and Warren counties, Dr. Frans attended Brown's Business College, at Galesburg, and the University of Illinois before entering the Chicago Veterinary College. Following his graduation in 1906, he entered practice at Monmouth. Dr. Frans was a member of Monmouth Lodge No. 37, A. F. & A. M., and had been a 32nd degree Mason. He is survived by his widow (née Mary E. Cook) and one daughter.

ANDREW ARTHUR BROWN

Dr. Andrew A. Brown, of Giltner, Neb., died at his home, April 17, 1937, following a stroke of apoplexy the day before his death.

Born at Letts, Iowa, December 27, 1870, Dr. Brown was graduated from the Kansas City Veterinary College with the class of 1908, of which he was president. Shortly thereafter he located at Giltner, Neb., where he continued in general practice until his death. He was a member of the Masonic Lodge of Giltner and a Shriner. He is survived by his widow, one son, one daughter, one sister and one brother.

E. C. J.

GEORGE MOSES

Dr. George Moses, of Amboy, Minn., died in Saint Barnabas Hospital, Minneapolis, on April 24, 1937, following an operation for chronic appendicitis and complications.

Born at Lake Crystal, Minn., on September 18, 1900, Dr. Moses attended local grade and high schools before entering Iowa State

College. Following his graduation in 1922, he located in Jackson, Minn., later going to Amboy to take over the practice of Dr. A. M. Oston (Iowa '22), when the latter decided to study human medicine. Dr. Moses was active in civic affairs having been elected a member of the local Board of Education in July, 1936, and president of the Amboy Parent-Teachers' Association two months later.

Dr. Moses joined the A. V. M. A. in 1930. He was a member of the Minnesota State Veterinary Medical Society and the Southwestern Minnesota Veterinary Medical Association. He was a Mason, an Odd Fellow and a Modern Woodman, as well as a member of the Sigma Sigma Fraternity. He is survived by his widow (née Louise Borchert), one daughter, one son, his mother, two sisters and one brother.

L. E. S.

CHARLES EDGAR POE

Dr. Charles E. Poe, of Hagerstown, Md., died suddenly at his home, May 2, 1937, following a heart attack. He was 69 years of age.

Born at Leitersburg, Md., Dr. Poe studied veterinary medicine at the Ontario Veterinary College. Following his graduation in 1902, he located at Hagerstown and was engaged in general practice there until 1917, when he entered state employ.

Dr. Poe joined the A. V. M. A. in 1902. He was a charter member of the Maryland State Veterinary Medical Association and a member of the Twelfth International Veterinary Congress. His fraternal affiliations included the Elks, the Knights of Pythias and the Odd Fellows. He is survived by his widow, two sons, one sister and two brothers.

M. W.

ELMER DEWITT NASH

Dr. Elmer D. Nash, of Helena, Mont., died May 5, 1937. His death came as a result of heart failure while he was actively engaged in the work of his profession.

Born at Dunlap, Iowa, December 16, 1869, Dr. Nash was graduated from the Grand Rapids Veterinary College in 1902 and was appointed Chief Deputy State Veterinary Surgeon on June 1 of that year, which position he held until December, 1930. For a period during the World War, Dr. Nash acted as State Veterinarian.

Dr. Nash joined the A. V. M. A. in 1915. He was a charter member of the Montana Veterinary Medical Association and was one of the old-timers in the veterinary profession in Montana. Under the direction of the late Dr. M. E. Knowles, who was then State Veterinarian, Dr. Nash was a pioneer in the work of state control of live stock diseases in Montana. He was known by all the veterinarians of the state and by the live stock owners as a true and loyal friend. He leaves his widow, one son and one daughter.

H. M.

LEONARD G. HART

Dr. Leonard G. Hart, of Chippewa Falls, Wis., died at Saint Joseph's Hospital, May 12, 1937, after an illness of about a week, caused by influenza and pneumonia.

Born at Eau Claire, Wis., December 22, 1895, Dr. Hart moved to Chippewa Falls with his parents when he was eight years old. He attended local grade and high schools before entering the Kansas City Veterinary College. Following his graduation in 1918, he returned to Chippewa Falls and engaged in general practice there until his death.

Dr. Hart joined the A. V. M. A. in 1919. He was a member of the Wisconsin Veterinary Medical Association and of the Wisconsin State Board of Veterinary Examiners. He is survived by his widow (née Ruth O'Neil), one son, his mother, two sisters and one brother.

J. S. H.

WESLEY D. McCOY

Dr. Wesley D. McCoy, colored veterinarian of Cassopolis, Mich., died in Oshtemo Sanitorium, May 20, 1937, at the age of 56. Death was due to tuberculosis. He was a graduate of Tuskegee Normal College and the Ontario Veterinary College, class of 1913. He had practiced at Cassopolis for 22 years. He is survived by his widow (née Perses Brice) and one son.

PERSONALS

BIRTH

To Dr. and Mrs. Irvin T. Reed, of Auburn, Ala., a son, Herbert Stanley, April 10, 1937.

PERSONALS

DR. J. E. DOBYNS (Ind. '13), is now practicing at Salem, Ind.

DR. J. B. WAY (K. C. V. C. '14), of Louisville, Ky., suffered a paralytic stroke on May 5.

DR. LINCOLN G. KUTSHER (U. P. '34), is now engaged in general practice at Stroudsburg, Pa.

DR. W. E. COOVER (Ont. '03), of Lexington, Ky., is veterinarian to the Kentucky Racing Commission.

DR. L. F. NEUENSCHWANDER (O. S. U. '32) has removed from Attica, Ohio, to Mechanicsburg, same state.

DR. ROBERT E. OAKS (O. S. U. '16) has located at Somerset, Ohio, where he will engage in general practice.

DR. J. R. KARR (O. S. U. '32), of Coshocton, Ohio, was confined to his home by illness for about two weeks in April.

DR. HERMAN KROGER (O. S. U. '25) has resigned as city veterinarian of Dayton, Ohio, to accept a position in Saint Louis, Mo.

DR. C. P. HACKETT (O. S. U. '36), formerly of Kinsman, Ohio, has opened a veterinary hospital at 6118 Market Street, Youngstown, Ohio.

DR. GEORGE F. MOTTELER (Wash. '36), of Pomeroy, Wash., has purchased the Idaho Veterinary Supply owned by Dr. T. A. Elliott, at Lewiston, Idaho.

DR. MAX STILES (Mich. '35), of Battle Creek, Mich., took a prominent part in the Battle Creek Community Fund 16th annual campaign the week of May 9.

DR. ARNOLD S. ROSENWALD (K. S. C. '36), of Winona, Minn., has accepted a position in the Department of Veterinary Medicine at Oregon State College, Corvallis.

DR. M. E. MOORE (O. S. U. '36), of Alexandria, Ind., has moved into a completely refurnished and modernized office and residence at 120 East Washington Street.

DR. CHARLES J. MULVEY (McGill '94), of Mooers, N. Y., was a patient at the Champlain Valley Hospital for about a month, following an injury received on March 21.

DR. J. J. KELLY (McK. '17), of Marshall, Minn., had the misfortune of having his office destroyed by fire recently, following an explosion in a restaurant in an adjoining building.

DR. E. G. PRINGLE (Mich. '32), of Goshen, Ind., addressed the April meeting of the Elkhart County 4-H Guernsey Calf Club. His topic was "My Experiences in Raising Guernsey Cattle."

DR. E. B. SIMONDS (U. S. C. V. S. '18), of Baltimore, Md., B. A. I. inspector-in-charge of tuberculosis eradication, has taken a leave of absence for six months in the interests of his health.

DR. ROY F. MILLER (K. S. C. '35) has resigned from the service of the U. S. Bureau of Animal Industry and has taken over the practice of the late Dr. J. Von H. Schantz, at Fairfield, Iowa.

DR. M. J. HUGGINS (Chi. '13), of Edwardsville, Ill., was reëlected Madison County Veterinarian by the Board of Supervisors at the May meeting, at an annual salary of $4,250. He has held the position for 14 years.

DR. W. N. ARMSTRONG (Ont. '94), of Concord, Mich., was reëlected president of the Jackson County Association of Justices of the Peace at the annual meeting held on April 21. The organization is the only one of its kind in Michigan.

DR. B. D. ROBBINS (Mich. '19), who has been in the service of the U. S. Bureau of Animal Industry for the past five years, has located at Holly, Mich., for general practice. He practiced at Grand Ledge, Mich., from 1919 until 1931.

DR. H. A. WHITE (Chi. '88), of Wyoming, Ill., was the subject of quite a newspaper article on March 10, marking fifty years of continuous service to his community. He practiced several years under a preceptor before completing his college course.

DR. C. C. HASTINGS (Chi. '13), of Williamsville, Ill., was the victim of a peculiar accident the latter part of April. While working with some cattle, one of them ran a horn through his foot between the toes. The injury was very painful but not serious.

DR. N. E. PEIGH (Chi. '20), of Hamlet, Ind., received three broken ribs when he was attacked by an angry bull while crossing a field on a farm near Knox, on April 20. He was saved from more extensive injuries by a dog, which drove the bull away.

DR. L. B. SWINGLEY (Chi. '09), of Oregon, Ill., received serious injuries of his right arm in an automobile accident the latter part of April. He was driving home at night and, in order to avoid hitting a calf on the road, he turned his car suddenly and crashed.

DR. DON R. COBURN (Mich. '24) has resigned as pathologist to the Game Division of the Michigan Department of Conservation, to accept a position with the U. S. Bureau of Biological Survey as associate veterinarian in charge of research in diseases of birds, with headquarters at the Bear River Migratory Refuge at Brigham, Utah.

DR. J. F. DEVINE (Amer. '95), of Goshen, N. Y., was among the speakers at the second annual dinner of the Springbrook Driving Association, held in Rhinebeck, N. Y., April 19. His talk covered the breeding of horses and cattle and was delivered in Dr. DeVine's characteristic style.

BUREAU TRANSFERS

DR. T. H. APPLEWHITE (Ind. '14), from Atlanta, Ga., to Jacksonville, Fla., as inspector in charge.

DR. W. C. DENDINGER (St. Jos. '16), from Augusta, Maine, to Atlanta, Ga., in charge of tuberculosis eradication.

DR. A. L. HIRLEMAN (Cin. '03), from Jacksonville, Fla., to Augusta, Maine, in charge of tuberculosis eradication.

DR. IRA W. EDWARDS (Chi. '11), from Nashville, Tenn., to Atlanta, Ga., on tuberculosis eradication.

DR. ORRIN W. NOYES (K. C. V. C. '04), from Evansville, Ind., to Leavenworth, Kan., in charge of meat inspection.

DR. TRUMAN W. COLE (Chi. '15), from Jacksonville, Fla., to Washington, D. C., as assistant chief of the Field Inspection Division.

DR. SYDNEY FOLSE (K. S. V. C. '09), from Bellows Falls, Vt., to Boston, Mass., on meat inspection.

DR. MALCOLM R. JOLLIE (O. S. U. '10), from Fort Branch, Ind., to Evansville, Ind., in charge of meat inspection.

DR. HARRY SEEVERS (Cin. '16), from Indianapolis, Ind., to Fort Branch, Ind., in charge of meat inspection.